The Royal London
Ophthalmic Hospital Reports.

VOL. XII. PART II. JULY, 1888.

Original Contributions.

CASES ILLUSTRATING QUESTIONS IN DIAGNOSIS AND TREATMENT.

By JONATHAN HUTCHINSON, F.R.S., LL.D.

No. I.—*Blow on the Eye causing numerous and long Lacerations of the Choroid. Subsequent Pigmentation of the Retina and Blindness.*

THE case of Mr. L., of Bradford, is of much interest in illustration of the results of a blow on the eye. It is remarkable also, because the blow was not inflicted by a hard body. When a boy at Christ's Hospital Mr. L. was struck on the right eye by a hollow india-rubber ball which had a hole in it. The effect of this hole was, he says, to allow the ball to collapse, and it is thus possible that it struck him edgewise. The blow gave him great pain, and, so far as he remembers, he was blind almost immediately afterwards. He was seen in the first instance by Mr. Stone, and subsequently by Mr. Power and Sir James Paget.

My examination of the eye was 12 or 15 years after the injury. It was then quite blind and diverged considerably. It was perfectly bright, moved easily, and I never saw a case in which it was more difficult to tell which eye the patient was using. Mr. L. said that this was a constant source of annoyance to him, and that in company, when addressing one person, he frequently

got a reply from another who was under the impression that he was looking at him.

The pupil was not larger than that of the other eye.

On examination with the ophthalmoscope I found a number of linear scars in the choroid. These were very conspicuous, and some of them very long; and it was remarkable that they almost all of them ran horizontally across the eye and not as usual in curved directions from above downwards.

One of them formed a Y with an acute angle, and one was so long that I did not succeed in finding either end. Some of them were small. I had only a short examination, as it was made to satisfy my own curiosity, and he had not consulted me about his eye. In addition to these scars in the choroid there were innumerable stellate patches of pigment in the retina. These were irregularly scattered over the whole fundus. They were not attended with any patches of choroidal atrophy, and in many instances they covered retinal vessels. The disc was not much paler than natural, but had a slightly hazy or gelatinous look. The central retinal vessels were not diminished in size, and in many places could be seen crossing the choroidal rents without impediment. It is to be understood that the rents were nowhere wide, but were for the most part mere lines. The media were quite clear, and there were no indications of choroidal disorganisation excepting the scars.

That pigmentation of the retina often follows blows is, I suppose, a well-known fact. I have seen several examples of it, some of them in syphilitic patients, in whom the question as to whether the changes were of a specific nature was very important. If pigmentation in such a case be in an advanced degree, and in one eye only, the history of injury should always be asked for. Sometimes after blows it is very extensive indeed. I have seen the retina in one case almost black from the confluence of stellate pigment accumulations after a blow. Much, no doubt, depends upon the complexion of the patient and the quantity of movable pigment present. Those of dark complexion are the most likely to show this condition. The same explanation applies to pigmented scars in the skin. Their darkness is an indication of temperament rather than of diathesis.

I do not for a moment believe that the pigment spots described above were the remains of blood extravasations, but

rather that they resulted from a very slight form of chronic inflammation, consequent on concussion.

No. II.—*On certain cases of Ulceration of the Cornea attended by very severe and paroxysmal Pain, and probably due to Gout.*

It is an open question whether there are any cases in which ulceration of the cornea is due to gout. We well know that many forms of iritis and sclerotitis, cyclitis, &c., occur in that connection, but I do not think that anyone has as yet associated corneal ulceration with that cause. I have not myself met with many cases in which I entertained any suspicion in that direction; but the following is one in which it seems very difficult to avoid it. It is not by any means the only one which I have seen.

Mr. B. B., a gentleman of about 60 years of age, has suffered repeatedly and severely from true gout. He has been laid up for several months at a time with attacks in the great toe and instep. He is a man of robust habit, and although a total abstainer as regards stimulants, is a large consumer of meat and fruit. He enjoys a good appetite and excellent digestion. In the end of January of the present year, whilst staying at Hastings, Mr. B. had an attack of inflammation of his *right* eye. It was attended by considerable pain, and he was told that there was an ulcer, but it soon got well. I did not see him during this illness. About a month later his *left* eye was attacked, and much more severely, and for this he came up to London and was under my care. There was marginal ulceration of the cornea at its upper border, and extending over at least a third of its circumference. There was great congestion of the conjunctiva and sclerotic, and some slight haze of the cornea beneath the ulcer. The ulcer looked very threatening, and, occurring in a man of 60, I was quite prepared to see it spread and produce disorganisation. The pain was, however, beyond anything which the state of the ulcer would have led me to expect, and there was the peculiar feature about it, suggestive of gout, that it was always much worse at night. On some occasions it was entirely absent in the daytime, and would then keep him awake the whole night; disappearing completely

in the morning. Mr. B. had had great experience of gout pains, and he said that the pain in his eye was exactly like what he was familiar with when he had gout in his toe. I prescribed for his attack on this hypothesis, putting him on low diet without any meat, and giving him alkalies, colchicum, and aconite. It may be briefly stated that under these measures the liability to pain entirely ceased and the eye was soon well. It is more than a year since the case was under care, and there has been no return of the disease.

No. III.—*On the paramount importance of Change of Climate in the Treatment of Cases of Inflammation of the Eyes from Inherited Gout.*

In the investigation of all forms of gout a clear distinction must be drawn between an inherited and an acquired tendency. In no department of the subject is this rule more important in its bearings than in respect of diseases of the eye. The forms of disease due to inheritance are wholly different from those which we see in the acquired disease, and, above all, they require a different treatment. They occur also for the most part at early periods of life, when acquired gout is rarely seen, and they very often show themselves in young females. In making these remarks, I by no means forget that there are but few cases of acquisition of gout in which there is not inheritance also. On the other hand, there are plenty where the inheritance is the sole cause of the disease, and in which the patient's personal life has not in the least conduced to it. In these latter the treatment by abstinence, alkalies, and colchicum would be most inappropriate.

I was recently asked in consultation in an obscure case of keratitis, in which the diagnosis lay between inherited gout and inherited syphilis, whether its establishment would make any difference in treatment. My friend suggested that in either case, as there was obvious inflammation, small doses of mercury and iodides with tonics would be the proper measures. Without controverting this proposition in its general bearing, I was yet inclined to attach great importance to another wholly different measure if the diagnosis of gout were made out. In all cases of inherited gout affecting the eye, whether serous iritis, cyclitis, relapsing iritis, sclerotitis, or any other special

pathological condition, the most efficient of all measures as regards cure is, I believe, a complete change of climate. It may be inferred from the mere fact of the initiation of the disease, that the place where the patient has been living has not wholly suited, and that a change is desirable. Any change to a drier or more elevated position is good, but if possible it should be out of England. The English climate with its cold and damp is very unfavourable in gout and rheumatism, and especially in those states of constitution which favour manifestations of it due to inherited peculiarity of tissues. I have published several cases in which those who were liable to that peculiar form of insidious iritis with vitreous opacities which I have ventured to associate with gout, have been quite cured by being sent abroad. I have seen several others in which the disease has persisted in spite of all other treatment in those who could not leave home.

I have recently had under care a young married lady of fragile development who inherited gout on both sides. She was the subject of iritis with corneal ulcers and some cyclitis in both eyes. The disease had alternated from one eye to the other, and for two years she had never been quite free. She had had much treatment under well skilled advice, and with only partial results. My advice that she should try a voyage to the Antipodes was resisted by a medical relation of hers, on the plea that she ought not to leave her surgeons as the eyes were still badly inflamed. I replied that this was precisely the reason why I wished her to go. She went, and the result was that she returned in a year with her eyes quite well, and having gained greatly in general health. The improvement began when she got into the tropics, but was only slow during a stay in Australia, where it was very hot. She did not get quite well until she reached Dunedin, New Zealand. The air of the latter place she described as suiting her case exactly, and there she wholly lost all remains of irritability in the eyes.

No. IV.—*A Case of Double Optic Neuritis in possible connection with Gout. Important facts as to Prognosis.*

A very interesting case of double, but unsymmetrical optic neuritis, in which there was a strong family history of gout, and no other cause discoverable, was brought to me by Dr.

Maurice of Marlboro'. On the first occasion I saw the patient in consultation with Dr. Hughlings Jackson also. This was in 1883. Dr. Jackson had carefully investigated the case in reference to any other cerebral symptoms, and could find none. When I saw the patient for a second time, more than two years later, they were still quite absent. The details of the case are the following:—Mr. H. F. M, æt. 18, is a strong, muscular young man, who has never suffered from any special illness. He has a large head, with rather heavy aspect, but never suffers from headaches, and never had convulsions. He sleeps well, and is said to be good-tempered. He is the seventh child in a family of eight, and all his brothers and sisters are healthy. He was brought up as a water drinker, but has recently taken beer. There is gout on both sides of his family. His paternal grandfather suffered very severely from it, and his mother has herself had some mild attacks. His eyesight had been failing for some months before I saw him; with his left he could only with difficulty read No. 12, but with the right he could make out 3 and $\frac{20}{50}$.

It was very difficult to be exact as to dates. He thought that his left eye had never been so strong as the right, but he felt certain that he could formerly see much better with it than now. His left disc was uniformly white, its edges well defined, and perfectly clean. The central vessels were of good size. The disc of the right eye was pale, and especially so towards the yellow spot. There were no evidences of recent inflammation. The only subjective symptom which I could get him to admit as having been present during the failure of his sight, was a feeling "as if his eyes were being poked in."

The above notes were taken in April, 1883. I saw him again with Dr. Maurice on August 31, 1885. His right eye had considerably improved, and he could now see $\frac{20}{20}$ and read No. 1. The left, however, had deteriorated, and he could barely count fingers. Both discs were uniformly pale, with perfectly clean edges, and central vessels of normal size. The left was much the whiter of the two. He was now 20 years of age, and Dr. Maurice confirmed his statement as to his having enjoyed excellent health since our last consultation.

No special treatment had been adopted, as we had been inclined to regard the symptoms as those of past disease. He had rested absolutely, and had done very little reading.

He said that he could read the newspaper through without its causing either eye-ache or head-ache. At this visit I learnt that one of his sisters had recently become the subject of phthisis, and further that one of his grandfathers had been epileptic. The changes which had taken place in vision between the two consultations made it certain that the morbid processes were not wholly at rest at the date of the first, and consequently that the conditions had not been present from childhood, as on the first occasion we had been half inclined to suspect. It is to be clearly admitted that the young man had never himself shown any symptoms of gout. If, however, we put aside the suggestion that his optic neuritis was of gouty origin, we have to admit that we are quite unable to suggest any cause for it, and further, that from first to last the case was wholly unattended by the more usual concomitants of that disease as met with in association with tumours and other intracranial lesion.

No. V.—*A Case of Optic Neuritis in a Young Man without Obvious Cause. Details of the condition eleven years afterwards.*

The case of a young man named Henry Falshaw was in some respects parallel to the one just mentioned. This man was under my care at the Ophthalmic Hospital about 10 years ago, when he came up from Sheffield on account of failure of sight. I did not see him again until recently, September 1st, 1885. During this long interval he had remained quite free from other symptoms of nerve disorder. His sight had got a little worse, but it still enabled him to go about, and with effort he could read large print. During the whole of the eleven years he said he had never been able to read a newspaper for pleasure, but by effort he could puzzle out a few lines. He had gained his living as "boots" at an hotel, next as ship's steward, and lastly as a warehouse porter. From each of these occupations he had been driven by his defective sight, yet he had not suffered from headache or any other ailment, and asserted that he was as capable of work as ever if he could only see. It did not appear that there had been any sudden increase in the defect, indeed it was by no means certain that he was much worse than was his state 11 years ago. I have not as yet been able to find my first notes of his case, but he assures me

that we took great interest in it. The conditions now present (1885) are: white atrophy of both discs without diminution of central vessels; one eye (the left) being much worse than the other. There is no history of true gout in his family, but his mother suffers from rheumatic gout.

When, as already stated in September, 1885, Henry Falshaw called on me, it was in the hope of assistance in getting some employment. He told me that since his attendance at Moorfields he had not had any illness with the exception of an attack of fever abroad. His sight had, however, become he thought somewhat worse. He appeared to be in excellent health, and said that he never suffered from headache, or any other symptom of nerve disturbance. I found both discs as white as paper, quite clean, and with central vessels of good size. The left was the most advanced in atrophy. With the left he could just see letters of 100 at 12″; with the right he could puzzle out words of large pica. He thought that he had got a little worse of late, but this did not appear very certain, for he admitted that he had never read a newspaper during the 11 years, and he could still by effort have managed to do so slowly.

He said that he was not ill in any way when his eyes first failed, and he believed that I was, when he was with me before, inclined to attribute his symptoms to smoking, but he added that I then told him that it was a very exceptional case.

No. VI.—*Loss of one Eye from Choroido-retinitis in early life. Syphilis possible. Gouty Congestion of the Damaged Eye. Diagnosis difficult between Gout and Syphilis.*

The case of Mr. G. is of sufficient interest to lead me to mention it, although it is impossible to feel quite certain as to the diagnosis.

His father suffered severely from gout, and he himself, although only 27, has had several definite attacks. There is also no doubt that his eye has recently (that is some years after the attack of inflammation which destroyed it) been liable to irritability and congestion in association with gout tendencies.

Mr. G. is at present blind in his right eye to the extent that he can only just count the bars of a window. With his back turned to the light he cannot count fingers, indeed he can barely

distinguish the hand. The history is that the eye began to fail when he was 14 years of age, and passed into its present condition in the course of two or three years.

It has suffered from a choroido-neuro-retinitis which has left the retina pigmented in delicate irregular streaks, dots, and patches, whilst the choroid is diffusely thinned and somewhat patched, and the margins of the disc ill-defined. The conditions approach somewhat to those of retinitis pigmentosa, but are on a much coarser pattern. There are a few small opacities in the vitreous, and a few delicate striæ in the periphery of the lens. The other eye is myopic but not otherwise diseased.

The state of the eye is exactly like that in a man named John W., in whom there was no doubt that syphilis was the cause. Mr. G. admits that he had some gonorrhœa in boyhood, and it is therefore possible that he had syphilis. His date of ætat 14, when the eye began to fail, may perhaps be a mistake. I incline on the whole to think it probable that his choroido-retinitis was syphilitic rather than gouty. The recent attacks of congestion have been, however, probably gouty. He has just had an acute and very painful inflammation of one finger, no doubt gouty.

The two cases (John W. and Mr. G.) are of much interest as instances of the loss of one eye by choroido-retinitis, the other remaining quite free, and the patient continuing for many years without any relapse of syphilitic symptoms.

I may just add that Mr. G. came to me on account of an attack of acute congestion of the lost eye, and that he suffered a few days later from severe pain in one of his finger joints. I have not the least doubt that these ailments were of gouty nature. The important question is as to whether gout ever causes choroiditis. On this point further observation is needed.

A CASE OF INTRA-ORBITAL HÆMORRHAGE AND OTHER EYE COMPLICATIONS IN CONNECTION WITH HÆMOPHILIA.

By Priestley Smith,

Ophthalmic Surgeon to the Queen's Hospital, Birmingham.

Hæmophilia is so rarely met with as a cause of difficulty or danger in ophthalmic practice, that it seems well to put on record the following case, in which, after a slight accident and a trivial operation, the life of the patient was for some days in considerable danger. For the notes of the case, and for unremitting care of the patient during the critical period, I am indebted to Mr. J. F. Blurton, M.B., my house surgeon.

F. K., a boy aged 17, a tube-drawer, came to the hospital on May 29th, 1888. Four days previously, while at work, he had been struck by a piece of brass tube just above the middle of the left eyebrow. The boy looked ill, and was suffering much pain. The left eyeball was pushed considerably forwards; the eyelids were swollen, tense, and slightly discoloured; the conjunctiva was injected and chemosed. The margin of the lower lid was stretched very tightly across the globe and slightly inverted, so that the lashes were in contact with the cornea, and at the line of pressure the cornea was ulcerated and infiltrating in a manner which threatened destruction unless the pressure were speedily relieved. Vision, so far as it could be tested, appeared to be reduced to perception of light. The fellow eye was sound. The case was regarded as one of free hæmorrhage into the orbit caused by the blow on the brow. The boy was admitted to the wards.

To relieve the pressure on the cornea the external canthus was freely divided with scissors and the conjunctiva scarified; the inversion of the lower lid was corrected with strapping and collodion; ice compresses were applied. On the following day slight bleeding occurred from the wound, but was readily stopped by pressure. During the next two days it recurred

several times and was less readily stopped; meanwhile the proptosis and swelling of the lids diminished and the condition of the cornea improved.

On June 3rd a careful examination of the boy was made in consultation with Mr. Jordan Lloyd, and he was found to be the subject of well-marked hæmophilia. There were many bruises on arms and legs, and swellings in both knees. Ergot and iron in a mixture and a free supply of green vegetables were ordered.

During the next twelve days the hæmorrhage recurred from two to five and even six times during every 24 hours. The patient became weak and much blanched; the pulse rate increased. The temperature was normal throughout. There was no blood in urine or fæces. Various means were employed to check the hæmorrhage. Before the hæmophilia was diagnosed the divided canthus was closed with two hare-lip pins and figure-of-eight sutures; this stopped the bleeding for about 24 hours, but the recurrence seemed worse than before. Later were used pressure with dry lint, with lint saturated with perchloride of iron solution, with Ruspini's styptic and collodion, with cocaine solution, solid perchloride of iron, tannin, iced water, hot water. The cocaine and the hot water appeared to have more influence over the hæmorrhage than any other applications. Internally he took in succession ergot and iron, hamamelis and dilute sulphuric acid, sulphate of magnesia, sulphuric acid, and arsenic. His diet consisted of a full allowance of meat and green vegetables, milk, and a minimum of other fluids.

On June 14th, 15 days after the operation, the hæmorrhage finally stopped, and the wound began to granulate well with slight suppuration. The patient rallied quickly from his extremely anæmic condition, and was discharged 10 days later with the cornea healed and vision almost completely restored. We learned afterwards that he had frequently suffered from obstinate epistaxis, and that if he cut his finger he found great difficulty in stopping the bleeding. No history of hæmophilia in the family could be obtained.

ON THE PROGNOSIS IN CHRONIC GLAUCOMA.

(Three Clinical Lectures delivered at the Hospital in June, 1888.)

By E. Nettleship.

Lecture I.

Gentlemen,

There is, as you are aware, considerable difference of opinion as to the best treatment for chronic glaucoma, when to operate, and if we do operate what operation to perform; and as to the results. It is to these points and to facts that lead up to them that I wish to ask your chief attention.

One obstacle to the formation of a trustworthy decision on any of the clinical features of this formidable disease is found in the long duration of some cases even if they be left entirely untreated; another in the doubt which occasionally surrounds the diagnosis of the disease in its earlier stages; another in the optical inconvenience which a large iridectomy often occasions when performed on an eye with good acuteness of sight;* another in the common belief that if iridectomy be done when the field is already very much contracted, the operation is apt to be quickly followed by a further loss of visual area, actual harm thereby resulting from the interference. Differences in the size and situation of the wound may also account for some of the varying results.

* Yet even as to this there are the widest differences; some persons—often persons on the fanciful side—are intolerably worried by the "rays" and "streamers" to which the coloboma often gives rise when isolated flames are looked at. On the other hand I have notes of two men in whom large iridectomies had been made *downwards and outwards* merely for the premonitory symptoms of glaucoma; nine years after operation, one of them, then 56, had V. $= \frac{6}{6}$ with the operated eye; and fifteen years after operation the other, then 44, also had $\frac{6}{6}$. Both were somewhat hypermetropic, but there was no measurable astigmatism in either of them.

In trying to come to any conclusion on this difficult and complex subject, it is of the first importance to have clear ideas about the natural history of the disease. I should like therefore to draw your attention first to some facts which should engender caution in drawing conclusions as to the effects of treatment in chronic glaucoma.

In most cases of chronic glaucoma, whether the developed disease have been preceded by a recognised premonitory stage or no, the changes and the symptoms progress steadily to total blindness in a time which is sometimes measured by months, but is more often perhaps a couple of years, less or more; in a great many cases the second eye suffers before very long; and when the stage of blindness has been reached the eyes usually remain quiet. It is also common knowledge that several of the factors in the glaucomatous process may vary much in relation to each other. I allude especially to the degree of tension, which in the quietest forms is sometimes never perceptibly increased, to the state of the pupil and anterior chamber, which may be almost, if not quite, natural; and to the changes at the optic nerve, where the depth of the excavation bears no constant relation either to the atrophic pallor of the disc or to the degree of tension of the eye. Many of these remarks are equally true of the acute forms of glaucoma.

One of the most important exceptions to these statements is found in the very wide range in the duration of different cases of chronic glaucoma when allowed to run their course; and another in the occasional occurrence of appearances at the disc very suggestive of glaucoma though without any symptoms or results of the disease. The latter observation shows the importance of any facts which tend to prove that chronic glaucoma begins, or may begin, rather in the optic nerve than in the ciliary region.*

* Brailey and Stilling have both adduced pathologico-anatomical evidence pointing in this direction.

I am convinced that chronic glaucoma is tolerably often much slower in its progress than we commonly think.* The most chronic case I have ever seen was that of—

Case 1.—Captain L. (P. 8, 103). This gentleman was told when he was about 25, by a high authority, that he had glaucoma in his right eye and would soon lose it unless he were operated on. This was about 1865, and the diagnosis was without doubt made after an ophthalmoscopic examination. He did not submit to operation. About five years later the same opinion was given again, and again he declined interference as he thought the eye was not getting worse. It was not until he was 45 that he took further advice, from myself (April, 1883), because this eye had lately become foggy, had seen halos around lights, and become subject to a sort of "dead pain." I found all the signs of well-marked chronic glaucoma, T. decidedly increased, anterior ciliary arteries enlarged and tortuous, disc rather gray and moderately cupped all over, the arteries showing marked spontaneous pulsation, pupil a little larger than the other, the eye M., 2·5 D., and sees when corrected $\frac{6}{9}$. F. moderately contracted all round, somewhat more so in the lower-inner quadrant. I again advised iridectomy, which he again deferred, preferring to use eserine. From this time the eye steadily failed, but did not become blind for another $3\frac{1}{2}$ years, that is, till he was about 49. Almost as soon as the eye was blind it became gradually congested and increasingly painful, not at all an ordinary conclusion of chronic glaucoma, and it was excised in Edinburgh, much inflamed and very painful, in December, 1886, about 22 years after the first diagnosis had been given. The other remained perfect throughout with $-2{\cdot}5$ s. and $-2{\cdot}5$ c., seeing $\frac{6}{6}$ as lately as February, 1887. This case is remarkable, not only for its extreme length but for the final onset of congestive symptoms and pain, the escape, apparently permanent, of the second eye, and the coincidence of myopia which the patient said had been present

* Some very chronic cases are mentioned in Vol. V of the German Handbook.

since he was quite a young man, if not earlier; still I see no reason for regarding the case as other than uncomplicated glaucoma simplex.

Case 2.—Another very slow case is that of Miss M. (P. 5, 175), which will be referred to again under malignant glaucoma. Miss M., at the age of 43, was nearly blind of very chronic glaucoma, which from the account she gave of previous opinions seemed to have begun as long as nine years before.

Case 3.—Mr. R. (P. 4, 88) was told that he had a "glaucomatous tendency" in L. when he was 70. At 76 he still saw $\frac{6}{18}$ though F. was contracted, T. +1, and the disc cupped and gray. His other eye had been nearly blind from the same cause since he was 60.

With such cases as these before us we cannot but be very cautious in inferring that a person whose discs show changes resembling those of glaucoma is really safe, however free he may be from the symptoms or other signs, of the disease. Of eyes showing such disc-changes I have seen several examples, usually in elderly persons, thus:—

Case 4.—Mrs. McR., 48 (P. 5, 118), showed in 1881 "both discs very suspiciously cupped, the R. especially so, ps. rather large;" and in 1885 "very suspicious looking cups, steep and almost up to the scleral edge. V. as before $\frac{6}{6}$, Hm. 0·75 D., T. quite n."

Case 5.—Mr. B. (P. 9, 57) has considerable M. In 1884 (æt. 57) complained that he could not read music quite so well as hitherto; V. corrected $= \frac{6}{18}$; no decided symptoms of glaucoma, but as o.ds. were cupped and pale and T. slightly increased, I feared chronic glaucoma. However, four years later (1888) I heard that his sight was still quite good.

That such suspicious appearances may precede the outbreak of rapid glaucoma is proved by—

Case 6.—Mrs. G., æt. 40 (P. 6, 183), whose R. in September, 1887, showed a central, large, very steep "physiological" cup (quite different from that in the L.). The eye was attacked by subacute glaucoma three months later.

Then we see instances of real glaucoma in one eye with a very deep, or large, normal depression in the other, as in—

Case 7.—Dr. —— (P. 12, 75) has had one eye excised for absolute simple glaucoma with retinal hæmorrhages and pain. For at least $2\frac{1}{2}$ years past the other eye has presented a pale disc cupped all over to the depth of about 1 mm., yet V. has remained $\frac{6}{6}$, F. of quite full size, and T. n. There were some hæmorrhages in this eye also for a time, but they became absorbed.

Case 8.—Edward S. (M. 1883, No. 1838) had subacute glaucoma of R. in 1883 cured by iridectomy; in August, 1885, the disc was noted as showing a large physiological cup, but with a good width of nerve substance at inner half; the whole disc too pale. The L. at same time with V. $\frac{6}{6}$ showed a deep physiological cup.

Case 9.—Mr. G. (P. 16, 8), æt. 50, has glaucoma simplex in L. with contraction of F., but in R. perfect F. and no symptoms. The discs, however, are much alike, showing a large deep cup on the y.s. side, leaving a broad belt of nerve substance on the nasal side.

Case 10.—John S., æt. 51 (T. 1879, No. 65), August, 1879, L. advanced chronic glaucoma. R. no glaucoma, V. $\frac{6}{9}$ with $-0{\cdot}75$ D., but "physiological cup large, circular, and very deep." May, 1882, "L. now quite blind. R. as before, and o.d. still showing a very steep, deep, circular cup, but nerve substance of good colour."

In these and other cases of the same kind it is quite likely that glaucomatous disease is beginning in the optic

nerve, and that the malady will after a time show itself by the customary symptoms.

Glaucoma is sometimes markedly hereditary. See Cases 21 and 22 in father and daughter, &c., and Cases 26 and 27 in father and son. This liability to descend in successive generations has often been noted, and as opportunities occur observations should be made on the state of the discs in the children of glaucomatous patients.

The occasional extreme chronicity of glaucoma precludes us as yet from saying with certainty whether the disease ever really stops short of blindness. Such arrest of progress, however, seems now and then to occur in the acute forms, as in the following cases:—

Case 11.—Richard B. (P. 2, 6), who had always been short-sighted, had an attack of redness of the eyes and fogginess of sight when he was 24. The sight got worse for about seven months, and then remained stationary. I saw him in 1873 when he was 26, with high M. (degree not measured), T. nearly n. V. 16 J. Discs atrophied and decidedly cupped. He wrote $2\frac{1}{2}$ years later that his sight was no worse. This would seem to have been a subacute inflammatory attack coming to a natural ending. The existence of M. is to be carefully noted.

Case 12.—Miss M. (P. 3, 94) when 49 (May, 1878) had "bad inflammation" in R. with severe pain in the head; the attack was acute; there was no sticky discharge. She considered that the sight was somewhat damaged by the attack. I saw her a year later and several times afterwards. V. of R. was always very nearly as good as L., $\frac{12}{12}$ partly, but the disc, carefully examined more than once, was quite different from L., being somewhat grey, and decidedly cupped up to the edge nearly if not quite all round. The L. disc was quite natural. I saw her as recently as November, 1886, when the eyes were unchanged.

Case 13.—Miss S. (P. 8, 16). In 1883 (æt. 49) R. had slight +T., o. d. of fair colour but well cupped all over, V. with

a weak − lens $= \frac{6}{6}$, and F. of full size. T. remained a little + for more than a year, but as lately as May, 1887, it was noted as n., and F. still full; indeed but for the cupped disc the idea of glaucoma could not have occurred during the last two or three years. The other disc has all along shown a large though shelving cup.

Case 32 may also be referred to here.

Later, in speaking of the curative effect of operation in chronic glaucoma, we shall have to control our conclusions by asking whether the operated eyes would have behaved differently had no treatment been employed?; and this enquiry may conveniently be answered here in connection with the natural history of the disease. Now, on examining some 25 cases of far advanced double chronic glaucoma in which no operation had been performed, I find that in almost all of them the character of the disease had been throughout exactly alike in the two eyes, and that in 10 of them it began practically at the same time in the two eyes, whilst in the remaining 15 there was an interval. Hence it seems fair to infer that where one eye has been operated on and the other not, a permanent or prolonged arrest of downward progress in the operated eye is attributable to the operation; and the same reasoning will apply when both eyes have undergone operation.

We may here mention some other points in the natural course of the disease.

Though the type is usually as just stated the same in the two eyes, a few exceptions occur. Thus C. (T. 1883, No. 52) had quiet glaucoma in the right in 1877, and an acute attack in the left in 1883. Mrs. B. (T. 1881, No. 35) lost her left from chronic glaucoma, and about two years later had the acute disease in the right.

Another important point is as to symmetry. This is not easy to settle, since our decision must depend upon how long a time we allow for the manifestation of disease in the second eye. We may ask first, what is the probability

that the second eye will escape? Of 66 cases of chronic uncomplicated glaucoma, I find that the second eye was free from disease in 31. In 26, or all but 5 of these, at least a year was known to have elapsed since the disease in the first eye; and in no less than 13 of these 26, five years or more (5—10 years) had elapsed without the second eye being attacked. It might be supposed that there was very little probability of the second eye suffering after so long a period as five years, yet, as we shall presently see, this is by no means the case, and we therefore cannot say that any specified interval insures to the second eye complete immunity from risk.

If we inquire next what is the probability that the second eye will suffer? we find that in 35 of the 66 available cases both eyes are known to have been attacked, and as this of course represents less than the truth, we shall probably be safe in saying that in something like two-thirds the disease is, sooner or later, symmetrical. Of the 35 cases known to be double, the second eye was attacked within a year of the first in 13, or about one-third. Of the remaining 22, the interval between the two eyes varied between one year and four years in 15, and between five years and ten years or more in 7; in only 2, however, was it more than six years, namely, nine years in 1, and ten years or more in 1. So far as these numbers go, they show that if the second eye escapes for one year (22 cases), there is about one chance in three that it will escape for five years or more (7 cases); so that if in an old or feeble person one eye is still free from chronic glaucoma a year or two after the other eye has become affected, we may sometimes hesitate to operate on the affected eye, since there is a fair probability that the unaffected eye may continue free during the patient's lifetime. It may be observed incidentally here that in acute glaucoma the interval between the two eyes is usually short, and that a very long interval is exceedingly uncommon. I have only one case of acute glaucoma with an interval as long as nine years between

the two eyes, against five chronic cases with this interval; and four of acute glaucoma with an interval of six years or more against about 16 chronic cases.* It is probable, therefore, that the more chronic the disease in the first eye the longer the second eye is likely to escape.

The Treatment of Chronic Glaucoma.

I do not think that eserine, except very rarely, can stop the progress of chronic glaucoma. It is, however, a valuable means of lessening the frequency and severity of the threatenings which often for a long time precede the establishment of the permanent disease, and thus it certainly helps to keep the disease at bay for a time. It also undoubtedly checks recurrent threatenings and fluctuations after operation. The following are good instances of the failure of eserine in chronic glaucoma:—

Case 14.—Caroline A. (M. 1885, No. 566) came in August, 1883, at the age of 47, with R. already much damaged by simple glaucoma with slight attacks of pain. It had been in progress at least four months, probably more, but had not been noticed at first as the other eye was perfect. When first seen, R. showed T. + 2, F. much contracted in the typical manner (see Fig. 1), o. d. deeply cupped, and V. (corrected for

Fig. 1.

* The actual relative frequency of the chronic and acute (including subacute) forms is about as 45 chronic to 50 acute and subacute (deduced from Priestley Smith's paper in Ophth. Soc. Transactions, vol. vi, p. 294, Table I).

H. 4·5 D) $\frac{6}{24}$, but still liable to variations and improved by eserine to $\frac{6}{18}$. Eserine (about 0·25 per cent.) was used regularly thrice daily for the next 18 months (till February, 1885), when as it was causing conjunctivitis it had to be discontinued. By this time, F. had become smaller, V. worse, and T. was still +. Iridectomy performed in April, 1885, had no decided effect.

Case 15.—Mr. B. (P. 8, 178), at the age of 37 had nearly lost his R. by chronic glaucoma, which had been established for at least a year, after several years of threatening; T. was now + 2, V. = 20 J. barely, o. d. very pale and cupped.

The L. was now (June, 1883), threatened; variations of V., slight increase of T., and a large, palish, shelving cup, but a. c. and p. were quite n., F. full size, and V. (with his mixed As. corrected) = $\frac{6}{9}$. He was a very punctilious patient, and used weak eserine quite regularly twice daily. At the end of six months' treatment there was no change, but after 14 months (end of July, 1884), V. had sunk to $\frac{6}{24}$, and F. was contracting on nasal side (Fig. 2). Upward sclerotomy was done on

Fig. 2.

August 1st, and eserine continued about once a day; V. improved to $\frac{6}{18}$ soon after operation, and (subject to frequent temporary variations), remained stationary for 18 months when (in March, 1886), variations became worse and more frequent,

V. was *barely* $\frac{6}{18}$ and F. rather more contracted. I now made a large iridectomy downwards with the result that he has so far (July, 1888, or $2\frac{1}{4}$ years) held his ground, V. remaining at $\frac{6}{18}$ with the proper lens; he has, however, still found the daily use of eserine necessary, and in fact has hardly ever missed using it since I first saw him five years ago. He also used eserine to the other nearly blind eye, and whether on this account or no, a little sight (letters of 20 J.) was still present as late as three years after he first came to me (I made no note at the latest examination).

Mr. B's. case is very instructive in some other respects.

He is very susceptible to the quality of his eserine, and discovered for himself that if the solution had become red from keeping or exposure, it always irritated the eye, and he could not use it. I always prescribed for him the salicylate, but it seemed not to have any better keeping powers than the sulphate.

His sight has, throughout, been subject to frequent fluctuations, and I think that two factors, muscular and vascular, can be recognised.

He is a small, thin, physically feeble man of most amiable disposition, but very excitable and fidgetty, engaged almost entirely in examining carefully the texture of the finer sorts of woollen broad-cloth. He said that before any operation was done, he could sometimes see his work quite well without any glasses (in spite of 4 D. of mixed As. chiefly H.), though at other times he could not do at all without them—ordinary varying accommodative asthenopia—the muscular factor.

Most of his variations, however, seemed to depend more directly upon variations in the blood supply to his eye (retina and ciliary muscle) than upon mere fatigue of accommodation, and his case furnishes one illustration amongst several that might be quoted of the fact that blood is always carried to the retina with somewhat more difficulty than to most other parts of the body. Thus, whilst on the whole Mr. B. saw best in the morning and worst in the evening (the rule in glaucoma), he sometimes observed dimness after breakfast and after mid-day dinner, *i.e.*, when there was a marked diversion of blood to

the digestive organs. He is always very emphatic on the effects of weather and temperature on his eyes; in cold weather he always feels well, his eyes are less liable to mist, and he uses eserine only about once a day; in hot weather his eyes are not so well, and he has to use eserine two or three times a day, whilst "remaining in a decidedly warm room for some time" always has a bad effect on his sight. It must be added that his cutaneous circulation is easily influenced, and that, either from agitation or exertion, he sweats most profusely. Thus, anything that dilates the capillaries of his skin or digestive tract affects his sight by interfering with the ocular blood supply, where the disadvantage under which the arteria centralis retinæ naturally labours is increased by a somewhat augmented intra-ocular tension, and perhaps by a diminished elasticity of the arterial coats.

A case illustrating the combined influence of increased intra-ocular tension and defective arterial supply due to aortic insufficiency, the whole resulting in extreme shrinking or obliteration of retinal arteries and obliteration with thickening of the peripheral retinal veins, is given (with an excellent Plate) in the 4th volume of the Ophthalmological Society's Transactions, p. 111.

Lecture 2.

We now pass to the results of operative treatment—Iridectomy and Sclerotomy—in chronic glaucoma.

One cannot usefully employ the strictly statistical method in this inquiry, because the disease is not common enough to supply a large material to a single observer, whilst the subject is, in this aspect of it, too complicated for collective investigation. I must, therefore, be content to give in detail a few illustrative examples followed by a short statement of each case that I have counted as successful.

Leaving out of sight exceptional or extreme varieties, and doubtful cases, like some of those narrated in the former lecture, we must, I think, in judging of the effects of treatment, subdivide our material, and note that what

we speak of generally as cases of chronic glaucoma are not all alike. We must, if we wish to be fairly precise, take notice of, and, indeed, if anything, accentuate the varieties.

We have, first, simple glaucoma, which runs its steady course to blindness without variations, fogs, rainbows, or pain. Some of these cases are, as we have seen and shall see, very slow indeed, whilst others are comparatively rapid. If only one eye be affected, the disease may, of course, very easily be overlooked by the patient; and this may occur when both eyes are attacked if, as is sometimes the case, in spite of considerable loss of visual area the central acuteness of sight continues good.

Case 16.—Wm. M., æt. 65 (T. 1886, No. 114), gradual failure of both eyes without any glaucomatous symptoms in either. L. getting dim four years, R. more recently. L. now no p. l., T. +2, a. c. shallow, lens hazy. R. with +5, 5 D. = 16 J., T. n., a. c. n., p. acts, o. d. gray, moderately cupped, f. contracted except down-out.

Case 17.—Mr. B., æt. 73 (P. 4, 119). L. went blind painlessly about 10 years ago, now T. +2, and the other signs of old glaucoma. R., failure recognised only about three months; no variations, no pain, no rainbows; different opinions have been given; now (June, 1880) T. +1, a. c. n. V. $\frac{12}{30}$ with +1·5 D., o. d. gray and distinctly cupped all over, f. shows qualitative defect in nasal part.

Case 18.—Miss R., æt. 37 (P. 9, 209), July, 1884, failure of L. recognised 3 or 4 years; R. considered to be perfect. Now, R., T. ? +, V. $\frac{6}{6}$, f. slightly contracted all round, o. d. pale, with well-marked glaucoma cup, p. n. L. T. ? +, V. $\frac{6}{6}$, yet f. contracted to within 5° of the centre in the lower, inner, and upper gradients, elsewhere 10—40° (Fig. 3), o. d. paler than R., with well-marked cup, p. n. Patient has advanced

aortic dilatation, and has had rheumatic fever twice. In July, a satisfactory sclerotomy was performed in each. In June,

FIG. 3.—Left field of Case 18 before operation.

FIG. 4.—The same field 3 years after operation (Sclerotomy).

1887, three years later, each eye was in the same state as on admission, seeing $\frac{6}{6}$ partly; f. in L. practically unaltered (Fig. 4), but in R. seemed to have become a little smaller at the nasal side.

The result of operation in this lady's left eye shows that glaucoma can sometimes be arrested with apparent finality by operation, even when the field is encroached upon almost up to the centre; and, as we shall see, the case does not stand alone in this respect.

CASE 19.—Mr. A., æt. 50 (P. 13, 198). A keen sportsman, had found for a year or two, perhaps more, that he was not shooting as well with the rifle as formerly, but he did not suspect till shortly before I saw him that this was due to a defect in the right eye, though on afterwards thinking it over he had little doubt that that was the true explanation. He never recognised any variations or fogginess, but simply found that on looking straight forward he could not see either print or distant objects so sharply as heretofore. When seen (November, 1886) R. showed T. $+\frac{1}{2}$, V. $\frac{6}{12}$, f. contracted at nasal side to 15° from centre, o. d. pale and cupped in the first degree, no pulsation, p. of same size as L. and acting well, a. c. rather shallow. L. V. $\frac{6}{6}$, normal in every way. R., large

iridectomy up; T. remained slightly more than L. for a considerable time. July, 1887, R. with −4 D. c., and 2 +D. s., $\frac{6}{12}$, f. perhaps a little smaller at nasal side. May, 1888, condition of R. unchanged, T. now quite n. L. n., as before.

Cases of the class just illustrated are generally thought to be particularly unfavourable for operation. But, besides the two just related, I have one—James B., æt. 69 (T. 1885, No. 239)—in which the eye has held for at least two years since operation, and another, Mary W., æt. 62, operated on at a late stage (T. 1885, No. 168) in which 2½ years have passed since sclerotomy without acuteness falling lower, though some further loss of field was noted at the last examination, and Cases 22 and 23 of still better success.

We have next the commoner cases, in which variations of sight caused by attacks of fogginess and rainbows form a conspicuous feature; and there may also be neuralgic pain in the eye, or in the corresponding temple, forehead, or side of the nose. Congestive, or so-called inflammatory attacks, also occur in some patients, and, in fact, this sub-group merges imperceptibly into that of genuine acute glaucoma. The note, however, of the whole group is a tendency to intermissions. These cases are relatively common; Case 15, already given, is a rather exaggerated instance, the following is better:—

Case 20.—Elizabeth P. (T. 1881, No. 25.) When 47 she had a foggy attack lasting some days, after an accident. Again, when 48, her vision was foggy for a time, and a few months later she became liable to see rainbows. When admitted, aged 49, Jan. 10, 1881, she said she had had several attacks of foggy vision each week lately, but had had no material pain at any time. Condition was the same in each eye, namely: T. +2 to 3; V. with +1 D. $\frac{6}{18}$; p. fairly active; a. c. shallow; f. contracted at upper inner quadrant to about 10° from

centre. Though T. was much and immediately reduced by eserine, the symptoms repeatedly returned, and three weeks later, Jan. 31, a large, double upward iridectomy was made. The fs. a fortnight later were recorded as a little more contracted at the upper inner quadrant than before—R. to about 5°, L. to about 3° from centre; this observation, however, was not repeated. The o. ds., of which there is no note previously, were somewhat pale; R. somewhat cupped, L. not cupped. In July, 1835, 4½ years after operation, V. of each $\frac{6}{12}$ with and without +1 D., very little As. Oph. o. ds. very pale all over, edges ragged, coats of veins rather thick and white, very slight, if any, cupping.

Case 21.—John R., 72. (T. 1878, p. 59.) A well-educated man. Said his sight had been definitely failing about eight months, but that for a year before his sight had often become misty for a short time if he got excited by reading anything interesting. Both eyes were alike. He had always been "short-sighted" (refraction not noted). When admitted, June, 1878, T. was barely +1 in each, V. words of 14 Jaeger slowly, o. ds. shelving slipper excavation, fs. contracted to from 3—10° in the down-in and up-out quadrants, down-out nearly full size but showing a scotoma in centre of the best part, 40° from fixation point. Iridectomy was done on R., and six weeks later on L.; R. having remained stationary and L. become worse during this time. He lived nine years after operation, and could see up to the last to read large print, sight remaining just as it was at the time of the operations.

Case 22.—Mrs. B. (daughter of the last case) (T. 1878, p. 59*a*), whose L. has always been very defective and useless from old convergent squint, became liable to rainbows soon after the death of her husband when she was 35. At first they came but seldom, but increasing in frequency during the last seven years, had lately come almost every day about 11 A.M. She had, of her own accord, taken to glasses with some relief before I saw her at the age of 42. I then found (July, 1878) R. H.m. 2·75 D. V. = $\frac{6}{12}$; T. and p. n.; f. not examined; no signs of glaucoma except that o. d., which was of full colour, showed a deep, bowl-

like excavation with a narrow zone of nerve-substance and a line of atrophied choroid all round. In Dec., 1884, $6\frac{1}{2}$ years, I heard that the eye was worse, but did not see her. Oct., 1885, $7\frac{1}{4}$ years, she came again, saying that a sort of curtain had gradually been coming over the sight of R. from above for some time past; with +3 D. the eye saw $\frac{6}{18}$, the upper part of f. was lost almost up to centre (Fig. 5), T. +1, o. d. gray and very deeply

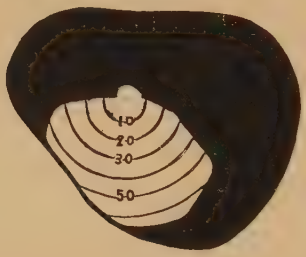

Fig. 5.

cupped, p. and a. c. n. (L. o. d. decidedly but not deeply cupped); a very large iridectomy was performed upwards in R. Oct., 1887 (two years after operation), R. keeps good, T. n., sub-conjunctival fistula at centre of wound. June, 1888, R. keeps good; +2 D. V. = $\frac{6}{18}$, and nearly $\frac{6}{12}$; wound still fistulous and T. less than L, o. d. very pale, and moderately cupped. L.—T. n., ? +, o. d. considerably cupped but not pale, veins full, f. full to hand test.

Her son has large, physiological cups. Her daughter has for some time complained of rainbows. Her father (previous Case) had chronic glaucoma, and one of her first cousins is said to have lost an eye from the same disease.

The late Mr. Critchett was, I think, accustomed to speak of certain rare cases as "acute simple glaucoma,"* which were characterised by *short, frequent* attacks of very severe dimness and tension without any pain or conges-

* Trans. of Ophth. Soc., vol. ii, p. 157.

tion, and which, owing to their high liability to internal hæmorrhage, were very unfavourable for operation. I mention the matter as there can be no doubt that Mr. Critchett had in his mind a definite clinical group, of which others have probably seen examples. But I am not sure that I have myself seen any cases conforming in all essentials with Mr. Critchett's description; Cases 20 and 30 are those that most nearly agree with it.

We now and then see cases which, though essentially chronic glaucoma, have some iritis, and for the present I will put such a case as the following, which is one of this kind, in a separate group.*

The cases, fortunately rare, of glaucoma occurring in eyes from which cataract has been removed should also be separately considered.

Case 23.—Mr. C., 45 (P. 8, 247), was seen in July, 1883, with rather quick but nearly quiet glaucoma, which had begun about four months before, in both eyes; the R. was, however, rather more advanced than L. When seen, July, 1883, R. T. +1, V. $\frac{6}{12}$, some fine dots on back of lower part of cornea, o. d. rather pale, excavated in saucer form all over, p. dilated from previous use of atropine, a. c. n., f. contracted to 5—10° everywhere except up-out, where it extends to about 60° (Fig. 6).

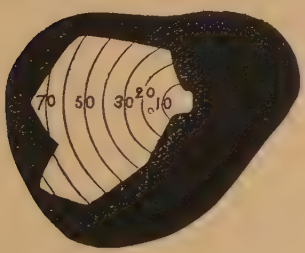

Fig. 7.

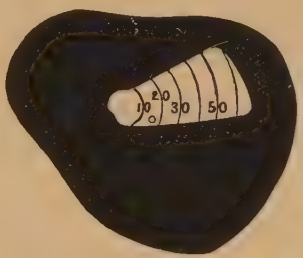

Fig. 6.

* I do not refer to the ordinary cases of so-called serous iritis or uveitis, in which there is often slight and usually transient increase of T. with deepening of anterior chamber.

L. V. $\frac{6}{6}$, T. ? +, f. of precisely the same type and degree of contraction near centre, but the remaining part broader (Fig. 7), no keratitis punctata; no atropine had been used.

With the traditional fear of operating with fields such as these, I temporised with eserine for nearly two months.

On Sept. 24, R. had T. still rather +, V. $\frac{6}{6}$ partly, dots on back of cornea gone, iris healthy, f. as before, p. still rather larger than L., still has rainbows. L. $\frac{6}{6}$ well, T. ? + (had been +1 a few days before), f. certainly smaller than before. "He thinks his eyes are about the same, but as T. at almost every visit remains +, and L. f. is diminishing, I now advise operation."

On October 4th, sclerotomy in each, converted into iridectomy in R. on account of prolapse. Both did well, but R. never saw so well after operation as before, chiefly, I think, owing to astigmatism.

April, 1888 ($4\frac{1}{2}$ years), R. with = +3 c. and −1 s. V. = $\frac{6}{18}$ well; L. V. $\frac{6}{6}$, Fs. tested by hand exactly the same as at last note, neither of the fields having enlarged at inner part; though central vision is perfect he has to go about very carefully and often knocks up against things, just as he did when first seen. Oph. shows the same now as on several occasions since the operation; o. ds. very pale, with saucer excavation, vessels about n. in L., arteries contracted in R.

This case I take to be a splendid instance of success from operating on chronic glaucoma with very bad fields.

You, of course, will not suppose that all operated cases have done as well, or been observed for as long as the ones I have just quoted, and, before concluding this part of the subject, I must therefore make some more general statement as to the results of my operations. Bearing in mind how tardy the disease may be, we shall do well to count as successes only cases that have been observed for, say, two years or more after the operation; and of such,

as you will readily understand, one cannot produce any large number.

Of 39 cases of chronic uncomplicated glaucoma operated upon with apparent success, which I have tabulated, I exclude one-half for various reasons, viz., 10 because less than two years has as yet elapsed since the operation,* 7 that were operated upon more than once, and were, therefore at best uncertain results,† and 2 following interstitial keratitis.‡ We have 20 left, including some that have been already narrated (Cases 18, 20, 21, 22, 23), and these are very shortly as follows :—

Case 24.—Mrs. W. Both began with some inflammatory symptoms in the spring of 1879, æt. 68. R. iridectomy six months after onset for steady but varying failure, V. being $\frac{6}{15}$, p. sluggish, F. somewhat contracted (Fig. 8). L. iridectomy 18 months after onset, V. being $\frac{6}{10}$, and F. smaller than in R. (Fig. 9). Nine years after operation on R. (eight years after L.) each eye as at date of operation. (Case I in Tables.)

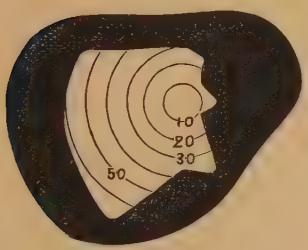

Fig. 9.

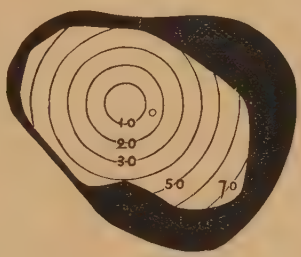

Fig. 8.

Case 25.—Mrs. B., æt. 73. L. nearly blind a year, T. +2,

* Nos. 10, 11, 18, 20, 23, 26, 27, 28, 32, and 37 in the tabulated statement above referred to. These tables I keep, but do not think it necessary to print.

† Nos. 2, 7, 8, 29, 35, 36, and 39.

‡ Nos. 31 and 33.

quiet. R. failing lately, V. $\frac{6}{18}$, F. typically contracted, T $+1\frac{1}{2}$, iridectomy; $3\frac{1}{2}$ years, condition the same. (No. 3 in ables.)

Case 26.—Mr. B., æt. 41. Duration uncertain; symptoms variable; V. in each $\frac{6}{18}$, T. +, ps. active, Fs. R. ? L. typical contraction. Sclerotomy in each; R. did well and got V. $\frac{6}{9}$, L. not so good, and never afterwards got $\frac{6}{60}$. $3\frac{1}{2}$ years, R. $\frac{6}{9}$; L. F. has become much smaller; o. ds. both pale and cupped. (4 in Tables.)

Case 27.—Mr. B., senior (father of last case). Iridectomy in each for chronic glaucoma, with partial cataract at 63; seven years later condition about the same; V. 20 J. Now had one cataract removed; aged 74, reported still to see about the same. (5 in Tables.)

Case 28.—Mr. J., R. iridectomy for advanced chronic glaucoma at 65; six years later V. the same as at operation = 19 J. (6 in Tables.)

Case 29.—James B., æt. 69. R. advanced chronic glaucoma. T. +2, V. $\frac{6}{18}$, o. d. cupped and pale. Six months later V. worse, not 20 J., F. contracted at inner part. Iridectomy. $2\frac{1}{4}$ years, still sees letters of 20 J., T. quite n. L. was more advanced than R., and lens became opaque after iridectomy; ? wounded. (14 in Tables.)

Case 30.—Miss B., æt. 46. L. "acute severe glaucoma simplex" coming on in an hour or so with very high tension and great loss of V., but neither congestion nor pain, after one year's threatening; iridectomy; four years later quite well. R. shrunk after operation for intermittent glaucoma at 37. This should, perhaps, not be counted as a chronic case. (16 in Tables.)

Case 31.—Ann B., æt. 56. Very advanced chronic painless

glaucoma both; R. p. l., T. +3; L. rather better; iridectomy both; $3\frac{1}{2}$ years, V. reported much the same.

Result of operations very late in glaucoma simplex. (19 in Tables.)

Case 32.—Francis E., acute attack in both, with remissions at 29, then better; at 34 failing again eight months; R. $\frac{6}{36}$, T. +1, F. n.; L. $\frac{6}{60}$, T. +3, F. much contracted at inner part. R. sclerotomy, L. iridectomy. Three years later, R. $\frac{6}{36}$, $\frac{6}{18}$ (corrected), F. and T. n. L. $\frac{6}{36}$, T. n., F. as before. (21 in Tables.)

Case 33.—Mary Ann P., æt. 45. R. failing with rainbows some months; V. $\frac{6}{24}$, T. +1, commencing cup, F. no note; L. almost absolute, T. +2, only p. l., failing one year. Iridectomy both; $6\frac{3}{4}$ years, R. the same; L. no p. l. (22 in Tables.)

Case 34.—Elizabeth C., æt. 49. Chronic glaucoma, with remissions and high tension two to three years. R. $\frac{6}{9}$, T. +2, deep cup, F. slightly contracted, a. c. very shallow. L. $\frac{6}{12}$ partly, T. +2—3, deep cup, F. much contracted, a. c. very shallow. T. in each considerably reduced by eserine. Iridectomy both; $3\frac{1}{2}$ years, reports V. keeps fairly well. (24 in Tables.)

Case 35.—Elizabeth B., æt. 70. R. failing six months, with pains and variations, $\frac{6}{24}$, F. extreme concentric contraction, T. +2, lessened by eserine, p. active to eserine, a. c. very shallow. Sclerotomy. $2\frac{1}{4}$ years, reports can still see a little with the operated eye.

This case cannot be called conclusive. (25 in Tables.)

Case 36.—Lucy D., æt. 52. R. failing some months, quietly; T. +2, much reduced by eserine, F. much contracted, no definite cupping, V., no note. Sclerotomy converted into

small iridectomy. Some months later, V. $\frac{6}{24}$; four years, sends a favourable report. (30 in Tables.)

Case 37.—Edmund H., æt. 26, began to get short-sighted; æt. 33, well-marked chronic glaucoma of both, about four years, L. rather worse; Fs. considerable contraction, deep cups, eserine not much action; V., R. with −3 D. $\frac{6}{24}$, L. with −3 D. not $\frac{6}{60}$. Iridectomy both; L. lost by immediate hemorrhage with severe pain; R. did well, and $3\frac{1}{2}$ years later saw $\frac{6}{12}$ with much contracted F., T. n. (34 in Tables.)

Case 38.—James P., at æt. 47, iridectomy in R. for early simple glaucoma with remissions; o. d. rather pale with a steep normal cup. Æt. 57 in same state, V. quite good. (38 in Tables.)

The following cases (in addition to Case 15 above), that were operated on more than once, have not been counted as successes, although in all of them the second operation seems to have stopped, or at least very much retarded, the disease:—

Case 39.—James A., æt. 43. R. iridectomy up for chronic glaucoma of about one year, exact state not known, effect lasted about 15 months, when patient thought V. worse. I now found V. $\frac{6}{60}$, F. contracted, T. ? +. Watched seven months, when F. more contracted and T. +1 ?. Sclerotomy down two years after iridectomy. $5\frac{1}{2}$ years after sclerotomy, V. the same. L. n., but o. d. suspicious. (8 in Tables.)

Case 40.—Sarah Ann M., 63. L., recent chronic glaucoma, ? duration, V. $\frac{6}{18}$, F. much contracted, very deep normal cup. Sclerotomy up; 10 days later, second sclerotomy down. One year after operation V. $\frac{6}{12}$. R. had absolute glaucoma with retinal

hemorrhages, and T. + 3; a few hours after iridectomy lens escaped; eye did badly and was excised. (27 in Tables.)

Case 41.—David E., æt. 63. L. threatenings for some time; V. $\frac{6}{6}$, T. n., F. n, o. d. suspicious; sclerotomy up. One year later $\frac{6}{12}$, two years $\frac{6}{12}$, some contraction of F., threatenings. $3\frac{1}{2}$ years $\frac{6}{60}$, F. smaller; iridectomy down with wound of lens. Lens was removed some time after and eye did well, getting $\frac{6}{36}$. Two years after iridectomy, or $5\frac{1}{2}$ after sclerotomy, keeps well. R. absolutely blind from simple glaucoma eight months before first admission. (29 in Tables.)

Case 42.—Mrs. Wiggins, æt. 53. R. acute glaucoma, did well with iridectomy. L. acute, following chronic glaucoma; iridectomy for the acute attack; eight days later second iridectomy; seven months later, lateral iridectomies; 13 months from first operation, sclerotomy up. One year after sclerotomy, two years after first iridectomy, V. 20 J., barely. Ten years after sclerotomy, æt. 63, sees fingers at 3 feet by moving the eye about. (39 in Tables.)

Case 43.—John P., æt. 67. R. failing six months; V. $\frac{6}{18}$, F. much contracted, T. slightly +. Iridectomy up, six months later sclerotomy up; three months after sclerotomy, second sclerotomy down; did badly, but gave no trouble; eight years after first operation quite blind and cornea dim. L. began about the same time as R. and thought to be worse; 16 J. barely when admitted; iridectomy up same date as R., nine months later imperfect sclerotomy up; four years later 20 J. ?; $6\frac{1}{2}$ years can still find his way about; eight years just sees large white objects in some parts of F. (33 in Tables.)

Case 44.—(35 in Tables), Lewis Lewis. Good result three to four years after two sclerotomies for chronic glaucoma with sharply contracted F.

As the cases quoted show there are different degrees of success amongst those classed together; and if we were content with a rather lower standard than I have taken,

we could increase our series by more than a dozen,* where the disease was stopped for a time, or decidedly retarded by the operation, or was apparently cured by a second operation.

By way of brief conclusion to this part of the subject, I may say that I have been agreeably surprised at the considerable proportion of good results after iridectomy or sclerotomy, or both operations, in chronic primary glaucoma, and that my faith in iridectomy for this disease, especially when performed early, is decidedly firmer than it was before I began to look into my cases.

It must be admitted, however, that there is much uncertainty, and that we have still much to learn before we shall be able to tell with any accuracy what influence various conditions in the patient and in the eye are likely to have on the result of operation. The chief of these conditions would be:—

The Stage of the Disease.—Though operation is no doubt much more likely to succeed if performed early in the disease than late, we have several cases where, with a late operation, the disease was permanently stopped with good result† (Cases 29, 21, 18, 24).

The state of the field affords no constant guide (Cases 23, 24, 21, 22, 18, and others).

Acuteness of Vision has no necessary relation to success or failure; permanent cure may follow operation on an eye with almost perfect acuteness though with very bad field (Cases 18 and 23), but may be just as marked in a case with defective acuteness but less extensive and less sharply defined loss of field (Cases 24, 21, 25, 32, 29, 34).

The Anterior Chamber, though often somewhat shallow,

* Nearly a dozen of the tabulated cases, Nos. 1, 3, 12, 14, 19, and 30 operated on once; and Nos. 2, 8, 29, 35, and 37 operated on more than once with partial success; and Cases 39, 40, 41, 42, 43, and 44 in the text operated on more than once with good success; in all 17.

† Even when the glaucoma is acute and operated upon early the result is not always good.

may be of good depth, as in Case 15 and others, or very shallow, as in Case 34.

Even *the Degree of Cupping and Pallor of the Disc* afford no constant guide to the future, as the successful Cases 18, 22, 26, and 34 show, in all of which there was considerable cupping.

The Tension, though sometimes scarcely increased, as in Case 18 and others, may be quite high, provided that it be intermittent; if tension continued very high, of course the case would not be chronic.

The State of the Pupil seems to me to furnish the best prognostic guide; in almost all of the successful cases it acted well to eserine, one of the best exceptions being in Case 37, but that patient, let it be noted, had considerable myopia.

Health and *Age* seem to exert a decided influence on success; at any rate, only two of the successful cases are noted as being conspicuously out of health, namely, Case 18, with advanced aortic dilatation, and Case 23, much over-worked and eating irregularly. On the other hand, amongst 11 cases that, without any apparent reason, did badly after operation, at least 6 are noted as being very cachectic in one way or another. As to age, in 26 successful cases (20 operated upon once, 6 operated upon twice) the patient was aged 45 or less in 8 (about $\frac{1}{3}$), and was over 60 in 9 (about $\frac{1}{3}$). Of the 11 unsuccessful cases above referred to, not one was below 47, and only 2 below 50, whilst 6 (at least $\frac{1}{2}$) were over 60.

Thus we may say that senile cachexia in its various forms is distinctly unfavourable to operation in chronic glaucoma, and that absence of senility and an active pupil are favourable points. The conclusion seems to me to be unavoidable that it is our clear duty to operate in chronic progressive glaucoma, and the earlier the better; and that even in advanced cases an operation should be performed unless there are special reasons (age, ill-health, complete and prolonged immunity of the other eye) rendering it

undesirable. If the disease seems to be stationary, and is in an early stage, and the patient can be watched and have his field measured at intervals of a few weeks, or later on, of some months, delay is always permissible and often desirable. In such circumstances eserine or pilocarpine should be used, but must not be strongly relied upon; and in no case of chronic glaucoma should treatment by eserine take the place of operation when it once becomes clear that the disease is advancing. Such advance may be shown either by decreasing area of the field or decreasing acuteness of central vision, but as either one of these symptoms may be much more marked than the other, the periodical examinations of the eye should include both.

Failure of Operation.—Many of the cases, whether chronic or acute, that fail unexpectedly do so owing to some unfavourable process going on at or near to the wound, and such unfavourable processes are more likely to come on when the patient is old or in bad health.

Some of these come under the heading of what has been described as "Malignant Glaucoma," tension not being relieved by the operation, and the anterior chamber not being re-formed. Displacement of the lens caused, in an eye with a weak suspensory ligament, by the suddenly reduced tension at the operation, probably accounts for some of these, and if we could in such cases remove the lens in its capsule with no worse consequence than the escape of some vitreous humour, it would be the best course to adopt. But it would seem that when the suspensory ligament is unsound, the intra-ocular blood-vessels are very often unsound too; at any rate, in the few cases of advanced glaucoma that I have had in which the lens has escaped in its capsule spontaneously within a few hours of the performance of a large iridectomy, the eye has been lost afterwards by hæmorrhage. Still there probably has been a case here and there in which a bold, skilful removal of the lens would have saved some sight; the

difficulty lies much more in recognising the case than in performing the manœuvre.

In other cases a chronic inflammation is set up in the wound leading, on the one hand, to slowly progressive haziness of the cornea, and, on the other, to permanent thickening and irritability at the scar*—a sort of sclero-keratitis.

As I have already said, I have not myself seen much, in chronic glaucoma, of that continued contraction of the field after operation, when the field was already a good deal contracted before, about which we hear so much in the books. I do not, however, think that we need be surprised that this should sometimes occur in acute as well as in chronic cases if the blood-vessels of the optic nerve and retina are unsound, as they frequently are in advanced glaucoma. The somewhat severe and prolonged congestion of the front of the eye that we often see after iridectomy for glaucoma may be taken as some index of the degree and duration of the congestion of the central retinal and choroidal vessels following on the rapidly diminished tension. A congestive œdema of the optic nerve thus set up might readily cause rapid increase of damage to nerve-fibres, most of which are already in a state of advanced degeneration; and especially so in a nerve where, as in advanced glaucoma, there is great increase of connective tissue. The occurrence of retinal hæmorrhages after the iridectomy, asserted by V. Gräfe to be so common, is mainly a sign of diseased vessels; I shall return to this subject in the next lecture in connection with hæmorrhagic glaucoma.

The result may also be bad, of course, from faulty performance, or faulty choice of operation, or for want of operating a second time. Probably all operators of experience have occasionally spoilt an eye by wounding the lens.

* I do not refer to the state of chronic porcellaneous-, pearly-, or milky-looking œdema of the conjunctiva which is sometimes seen over the wound, and which is certainly often, probably always, an evidence that the scleral wound is leaking, and is therefore so far a good sign.

With respect to the choice of operation, I can only say that I have come back to iridectomy in chronic glaucoma after a tolerably extensive trial of sclerotomy. The sclerotomy I performed was always made sub-conjunctivally with a Graefe's knife, and always with the preservation of a narrow bridge of sclerotic. I operated on quite 50 cases, and therefore got over the initial difficulties of the proceeding. The results were for a time very pleasing, but as time went on I found that some of the cases gradually went down hill, and that others had to undergo a second operation, usually iridectomy. Although I have had a few very brilliant cases of permanent success after sclerotomy (Cases 18, 23, 26, 32, 44), I have not been able to make out why these should have succeeded, and I therefore never feel able to say on looking at a case before operation whether sclerotomy is likely to succeed or not. Sclerotomy being a more difficult operation than iridectomy will always be fraught with more risk. For these reasons and from having become gradually convinced that iridectomy often gives excellent results in chronic glaucoma, even when the field is much contracted, and that the optical inconvenience of iridectomy can in great measure be remedied by the careful correction of the astigmatism, I feel myself unlikely to revert to sub-conjunctival sclerotomy as a primary operation. But I shall still be prepared to perform it when iridectomy has failed, placing it then on the opposite side to the iridectomy. I should say, however, that I have never made use of sclerotomy as it was originally performed with the lance-keratome by Quaglino, and is still employed I believe by Snellen and others; nor have I ever systematically repeated the section in two or more opposite positions as has been advocated by some of the French surgeons. I have, however, no doubt that such multiple incisions are surgically correct, and that we sometimes fail to arrest glaucoma for want of a timely second operation.

(*To be concluded.*)

ON THE NATURE OF LIGHT-PERCIPIENT ORGANS AND OF LIGHT- AND COLOUR-PERCEPTION.

By R. Marcus Gunn,

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In endeavouring to determine what ocular structures are certainly to be regarded as light-percipient,* we are met by the difficulty that sensibility to light may exist without any recognisable eye or eye-spot whatever being present. Indeed, a reaction or movement determined by light is sometimes observed in animals destitute, not only of eyes, but of any nervous system; and there are also numerous instances of its effect on the movement of vegetable organs and organisms.

Stahl,† for example, found that in *Closterium*, a form of *Desmid*, light induces the cells to alter their position according to its direction. When placed in a glass vessel, illuminated only from one side, the cells attach themselves to the glass so that their long axes coincide with the direction of the incident light. On altering the direction of the light the position of the cell changes in a corresponding manner with considerable rapidity.

This property of *phototaxis*, by which cells come to arrange themselves with their long axes parallel to the

* It may be contended that "light-sentient" would be a more correct term to apply to the end-organs of the visual apparatus. I have avoided the use of that term, however, on account of the difficulty that would often necessarily arise in distinguishing between cells or organs that are sensitive to or acted on by light, and those that are not only sensitive to light but inseparably connected with the visual function. Thus all young vegetable and even perhaps animal cells may be spoken of broadly as light-sentient in so far as they are acted on by light, but I am limiting myself, in the case of animals, to the consideration of such light-impressions as are capable of being appreciated by a visual nervous centre.

† "On the Action of Light on a Desmid." Reference in Journ. Roy. Micro. Soc., 1879, p. 37. Original article in Verh. Phys. Med. Gesellsch., Wurzburg, xiv, No. 7, and "Hedwigia," xviii, 1879, p. 100.

direction of the incident light, is found to have a very wide range of influence on the growth and behaviour of plants. Thus it determines the direction of movement of the free swimming zoospores, which arrange themselves so that their long axes are parallel to the light rays, and invariably travel directly away from, or directly towards the light, never in a cross direction. The end provided with fine protoplasmic processes or cilia always goes first, so that when the zoospore ultimately comes to rest, its ciliated end becomes the base of the germinating plant.

According to the direction of movement being towards, or away from, the source of light, we have what vegetable physiologists call respectively *positive* or *negative* phototaxis.

Similarly, light has an action on the radial organs of growing and of mature plants, by reason of this same property of phototaxis, or its parallel *heliotropism*. One of the most striking instances of the effect of light on the position assumed by vegetable organs is afforded by the way in which the movement of the young shoots of seedling plants is influenced by illumination. These always turn directly towards or directly away from the source of light, and the effect is induced by an illumination so feeble as to be practically indistinguishable by the human eye. In the case of most plants the movement is towards the light. Darwin,* to whose careful observations we owe our chief knowledge regarding this action of light on young plants, found that the curvature of the whole shoot was determined by the direction of the illumination of its upper extremity. "It can hardly fail," he says, "to be of service to seedlings, by aiding them to find the shortest path to the light, on nearly the same principle that the eyes of most of the lower crawling animals are seated at the anterior ends of their bodies." "We should bear in mind," he continues further on, "that the power of bending to the light is highly beneficial to

* "The Power of Movement of Plants," pp. 484 *et seq.*

most plants. There is therefore no improbability in this power having been specially acquired. In several respects light seems to act on the tissues of plants in nearly the same manner as it does on the nervous system of animals."

The arguments adduced by Darwin in favour of this latter statement may be summarised as follows:—

(*a.*) The effect is transmitted from one part to another, in seedlings as in animals.

(*b.*) An animal may be excited to move by a very small amount of light, and so also can certain plants.

(*c.*) The retina, after being stimulated by a bright light, feels the effect for some time; so in like manner a seedling continued to bend for nearly half an hour towards the side which had been illuminated.

(*d.*) The retina does not readily perceive a dim light after it has been exposed to a bright one; so plants which had been kept in the daylight during the previous day and morning, did not move so soon towards an obscure lateral light as did others which had been kept in complete darkness.

In all experiments of this nature, I need hardly say, the observers have been careful to discriminate between the effects of light and those of heat.

These effects of light on plants are also to be distinguished from the *chemical* changes produced in them by this agent, such as the green coloration of chlorophyll, the decomposition of carbon dioxide, and the formation of starch, sugar and oil. These latter are produced chiefly or solely by the rays of low refrangibility, viz., the red, orange, yellow and green. *Protoplasmic movement*, on the other hand, *and phototaxis, are produced chiefly* or *solely by the rays of high refrangibility*, viz., the blue, violet and ultra-violet, the less refrangible rays acting in this respect like darkness.

As further examples of the action of light on plant-movement I may mention observations of Baranetzky and Engelmann. The former* found that the plasmodes of *Aethalium*, a fungoid growth affecting tan, creep along the surface in the dark, but withdraw into the deep parts of the bark on exposure to a bright light. They have long, thin ramifications in the dark, while in the light they only develop short, thick processes.

In a large *bacterium*, to which he gave the specific name of *photometricum*, Engelmann† found a decided reaction to light. It is motionless in darkness, but moves actively on exposure to light for a few minutes. On prolonged exposure, the amount of light remaining the same, the movements again cease: the length of the period of irritability varies directly with the length of time the bacterium has been previously kept in darkness. Viewed individually they appear colourless, but large collections of them appear of a reddish colour, and the movement is best excited by the part of the spectrum corresponding to the absorbed rays.

Most forms of contractile *animal* protoplasm seem not to be acted on by light, but Engelmann‡ observed a peculiar effect of light on a very low fresh-water amœboid organism (*Pelomyxa palustris*). On shading it with the hand, the protoplasmic movement becomes more lively, while on *sudden* illumination the body almost immediately contracts into a round lump as after an electric shock. No irritation is produced by a gradual increase of light, nor by darkness suddenly produced after prolonged illumination.

* Baranétzky. "Influence de la lumière sur les plasmodia des Myxomycètes." Mém. Soc. des Sc. Nat., Cherbourg, xix, 1875, p. 321.

† "Bacterium photometricum." Ein Beitrag zur vergleichenden Physiol. der Licht- und Farbensinne. Onderzoek. in het. Phys. Laborat. te Utrecht, vii, 2, p. 252.

‡ "Ueber Reizung contraktilen Protoplasmas durch plötzliche Beleuchtung." Onderzoek. ged. in het. Physiol. Labor. der Utrechtsche Hoogeschool, v, p. 181. See also Archiv f. d. ges. Physiol., xix, p. 1; and Quart. Journ. Micro. Soc., vol. xxiv.

Tremblay,* again, found that the *hydra*, although destitute of any special organ of sight, is attracted towards the light, and he satisfied himself that this attraction was not due to the heat waves.

Effects such as I have just mentioned, induced by light in very low animal organisms, cannot be considered as examples of true visual power, but rather merely as simple protoplasmic changes produced by light, or at most as instances of one protoplasmic change determining another without consciousness. The importance of these observations on the action of light on low organisms and on cell protoplasm will be apparent later, when we come to consider the nature of the changes occurring in the retina on stimulation, and a full description of the known vital phenomena accompanying the light action will be deferred till then.

Different observers have proved the existence of a light sense in higher eyeless animal forms. The lower acari and the eyeless larvæ of certain dipterous insects, for example, exhibit a fondness for light; while others, which like darkness, are put to flight by light, even by that of the moon. The common earthworm, also, though destitute of eyes, has been shown by Hoffmeister to be extremely sensitive to light, and his observations have been confirmed by Darwin. The latter authority says, "sometimes . . . as soon as the light fell on them, they withdrew into their burrows with almost instantaneous rapidity. . . . When they did not withdraw instantly, they often raised the anterior tapering ends of their bodies from the ground, as if their attention was aroused, or as if surprise was felt. . . . In all cases the duration of the light, unless extremely feeble, made a great difference in the result; for worms left exposed before a paraffin lamp or candle invariably retreated into their burrows within 5—15 minutes. From the foregoing facts," he adds, "it is evident that light affects worms by its intensity and by

* "Mémoires pour servir à l'historie d'un sens de Polypes," t. 1, p. 137.

its duration. It is only the anterior extremity of the body, where the cerebral ganglia lie, which is affected by light. . . . We must suppose," Darwin continues, "that the light passes through their skins, and in some manner affects their cerebral ganglia." He found, further, that they were apparently less sensitive to moderate radiant heat than to a bright light. "The light from a candle, concentrated by a lens, and passing through a sheet of glass, which would intercept most of the heat-rays, generally caused much more rapid retreat than did the heated poker."*

With exceptions such as those we have noted, however, some form of eye or eye-spot is always met with in association with sensibility to light. The amount of differentiation of the structure having this function varies very greatly, but the presence of pigment in connection with it is invariable normally. Exceptionally, it is true, we find albino individuals having eyes without retinal pigment, but in them the light-percipient organs are otherwise well developed,—notwithstanding which, their vision is never so acute as in similar animals with pigmented eyes. We may lay it down as a law that the lower the type of eye the more necessary is the presence of pigment to its function, and we are thus prepared for a further generalisation, viz.: Whenever in any low animal organism a definite pigment spot is found, constant in its occurrence and in its position, and without any other obvious function, we are justified in regarding it as light-percipient. This conclusion is rendered practically certain if the pigment-spot be situated at the anterior end of the body of a freely moving animal, in connection with its nervous system, and if it be symmetrically disposed with any other pigment spots present that are apparently similar in function.

We must not be deceived, however, by mere symmetry. Thus, for example, a peculiar exceptional development of

* "Vegetable Mould and Earthworms," pp. 19 *et seq.*

pigment, in the form of two bilaterally symmetrical deposits, occurs in *Cephalodiscus*, a very singular new form brought home by H.M.S. "Challenger," described by Professor McIntosh, and placed by him provisionally among the Polyzoa. Through this gentleman's kindness, specimens were sent me for examination. At first sight, these pigment-spots suggest the presence of eyes, but further examination showed conclusively that they could not be credited with any light-percipient function. The animal is sedentary, and the pigmented areas are not in direct connection with its nervous system. I believe they have quite another function, connected with light it is true, viz., that of developing heat in the oviducts on which they are placed.

In certain *Infusoria* among the *Protozoa*, we first meet with pigment-spots that seem to serve as light-percipient organs. These spots are confined to freely moving genera, and always occur at the anterior end of the body, *i.e.*, at the end which goes in advance. As these animals are unicellular organisms, there is no specialised nervous system, and the light impression produced upon the eye-spot is probably communicated directly to the surrounding protoplasm. Though these animals are necessarily destitute of any specialisation of organs for sensation, motion, digestion, &c., the whole cell may be regarded as serving by turns all these functions, sometimes moving as a whole for prehension of its food, sometimes acting as a digestive system, and also always capable of receiving impressions from without such as we *generally* find associated with the presence of distinct nerve-cells. In one of the best known of these eyed Infusoria, the *Euglena*, the eye-spot consists of plasma having a net-like arrangement, in the meshes of which are situated tiny drops of an oily red substance allied to chlorophyll.* Excep-

* Klebs. "Ueber die Organisation einiger Flagellatengruppen in ihre Beziehungen zu Algen u. Infusorien." Unters. Bot. Inst., Tübingen., 1883, p. 233. (Ref. in Zool. Jahrb. f. 1883.)

tionally, as in *Erythropsis agilis*, there is in addition a strongly refractive, colourless, conical crystalline body lying upon the pigment, doubtless intensifying the action of light by concentration. It is probable that the principle of phototaxis determines the position of the eye-spot at the anterior end of the body in such Infusoria. The elongated form of the mature, as compared with the more rounded shape of the young individual, may well be the result of movement, in as far as the end provided with the eye-spot always goes in advance, and the tendency of mere friction will therefore be to lengthen the cell, and to make the posterior extremity fine and tail-like.

Among the *Hydrozoa* we first meet with visual organs consisting of *modified epithelial cells*, some of the latter being always conspicuous from the development of pigment within them.* We meet with many variations between simple pigment-spots on the marginal bodies and a highly developed eye on the *camera obscura* type, provided with a lens, such as is found in *Charybdea marsupialis*.

It is not my object in this short paper to describe at length the individual features of the sight-cells n different animals, but merely to draw broad conclusions as to the conditions common to all.

I. *In all the invertebrate groups the light-percipient cells are developed from the ectoderm*, as in the *Hydrozoa*. Sometimes this takes place by a very simple and obvious modification of the epithelium, as in the rudimentary eyes of the starfish, the lower worms, the larva of *Dytiscus* (among *Arthropods*), and in the *Patella* and *Arca Noæ* (among *Molluscs*). In highly developed eyes, *e.g.*, that of

* "Das Nervensystem u. die Sinnesorgane der Medusen." O. u. R. Hertwig, 1878. Pl. viii, figs. 9, 10, 15, and Pl. ix, figs. 5 and 13.

"Die Medusen (Physiol. u. Morphol. untersucht) auf ihr Nervensystem." Eimer, 1878, pp. 163–4, and Pl. iv; figs. 1, 3, 13, 14, &c.

"Bemerkungen über die Randkörper der Medusen." Gegenbaur, Müller's Arch. f. Anat. u. Phys., 1856, p. 231 and Pl. ix.

"Die Sehorgane der Thiere." Carrière, 1885, pp. 90–95, and fig. 73.

Alciope (among *Vermes*), the stemma of the Spider, and the compound eye of Insects and Crustaceans (among *Arthropods*), and the Cuttlefish (*Mollusca*) the epithelial origin is not so easily traced, and must be followed through intermediate forms.

In the case of the compound arthropodous eye, there is still some difference of opinion as to the manner in which the different parts have been developed, and as to how they are to be interpreted. I may, therefore, endeavour very shortly to trace the divergences of structure from the primitive form, as met with in the larva of *Dytiscus*, the eye of which has been described by Grenacher.* Here there is a single cornea-lens, produced by a thickening of the chitinous cuticle; behind this is a single layer of cells, continuous with those of the hypodermis or surface-epithelium. The cells at the periphery of the eye are long, and bend obliquely forward and inward towards the optic axis. The more median cells, on the other hand, project forward toward the lens, and become differentiated into nerve-end cells, each developing a rod anteriorly and ending posteriorly in a nerve-fibre. Pigment granules are formed throughout the whole length of the cells next the hypodermis, but only in the posterior half of the peripheral cells which reach the lens or which meet in the optic axis: the anterior part of these remains clear so as to permit the passage of light. These cells may consequently be said to have a double function, viz., to shut off lateral light by their bases, and to act as a vitreous body in their anterior transparent extremities. Pigment granules also develop to some degree throughout the whole length of the spindle-shaped, nervous or retinal cells.

* To the latter authority, and to the work of Ray Lankester and Bourne, I am chiefly indebted for my data.

Grenacher, "Untersuch. über das Sehorgan der Arthropoda insbesondere der Spinnen, Insecten und Crustaceen." Göttingen, 1879.

Ray Lankester and A. G. Bourne. "The Minute Structure of the Lateral and the Central Eyes of Scorpio and of Limulus." Qu. Jour. Micr. Sc., 1883, p. 177.

This simple form of eyelet may be modified in several directions, which may be characterised as—Grouping of the eyes as a whole; Grouping of retinal cells; and Development of a distinct vitreous layer.

A. Several similarly constructed ocelli may be grouped so closely as to touch one another, so that we get a very simple form of what may be called a composite eye. This condition exists in *Iulus* among the *Myriapoda*.

B. The vitreous cells may be further developed and displaced from their *lateral* into an *anterior* position as regards the retinal cells. Here they form a complete layer of clear "vitreous" cells. As examples of this form we have the stemmata of insects and the eyes of the spiders.

B′. A further modification may result from grouping of several such eyelets, so that we get another form of composite eye, as in *Lithobius* and *Scolopendra* among the Myriapoda.

C. The rods, instead of being developed *in front* of the retinal cells, are often modified so as to form hard ridges on one side, in front of the nucleus. The ridges of two or more adjacent cells now adhere together to form what has been called a rhabdom. From this change we get an organ such as is found as the lateral eye of the *Scorpion*, according to the description given by Ray Lankester and Bourne.

C′. Several of these, again, may be grouped together, as in the lateral eye of *Limulus* (or King Crab), forming a simple variety of the true compound eye. Perhaps we may place here also the more closely grouped compound Pseudoconous eyes of certain insects, *e.g.*, *Musca*.

D. If now we get a combination of the two chief mentioned modifications, viz., the formation of a distinct layer of vitreous, and also the formation of rhabdomes, we have the condition found in the central eye of the *Scorpion* and of *Limulus*.

D′. Close juxtaposition of such ocelli gives us the fully developed compound eye of *Insects* and *Crustaceans*.

In the typical *facetted* or *compound* eye we have, corresponding to each facet, a transparent crystalline cone or its equivalent next the cornea-lens, and behind this the retinula, or group of modified epithelial cells, with the central rhabdom. The broad end of the crystalline cone is applied to the inner surface of the cornea-lens; its narrow end is turned towards the outer extremity of the retinula. The latter invariably consists of a definite group of vertically elongated cells, having in its centre the clear rod or rhabdom. A pigmented sheath formed of several pigment-cells encloses each "element" more or less completely, separating it from its neighbours; some of these are in very close relation with and dip in between the free sides of the cells of the retinula. The inner extremity of each cell of the retinula is connected with a fine terminal nerve-filament, which immediately pierces a perforated basilar membrane. The true retina of Insects and Crustaceans, however, does not end at the basilar membrane, as was formerly described. According to Lowne, indeed, it is situated entirely behind this membrane. It would be more correct to regard the retina as consisting of the retinular cells, along with part at least of the structures known as the optic ganglia, lying between the basilar membrane and the optic nerve.

It will be out of place here to enter into the various modifications of this structure which may be met with at different stages of development of the same animal, or in different animals. We have chiefly to deal with the cells constituting the retinula, and they are always formed on the same plan. There is little doubt from their homology, as illustrated by the very brief sketch that I have given of the different forms of arthropod eyes, and from their anatomical relations, that the cells of the retinula are really the light-percipient organs. Thus they are placed so that light may penetrate to them; they are in close relation with pigment cells, and are themselves slightly pigmented; and they are in immediate connection with nerve-filaments.

The function of the central rhabdom I believe to be mainly that of affording support to the cells grouped around it, and secondarily, presumably, that of each rhabdomere transmitting light to its cell.

The pigment-cells are usually found in three series:—

(1.) Ensheathing the crystalline cone, preventing the entrance of lateral light, and the escape of light once admitted.

(2.) Pigment-cells situated on the outer region of the retinulæ, and sending processes between the individual sight-cells.

(3.) Similar pigment-cells resting upon the basilar membrane round each retinula. These two latter prevent light passing from one retinula to another by reflection, and probably serve a visual function in addition.

In many of the Crustacea there is a spindle-shaped thickening of the retinula, transversely banded with pigment, situated near the basilar membrane.

II. *The inner end of the sight-cell in invertebrates is either continued as a nerve-fibre, or is connected immediately with a ganglion cell, and ultimately with a nerve-fibre.*

III. *The outer extremity of the cell is, in all the higher forms of eye, with the exception of some of those met with in arthropods, covered with a cuticular structure in the form of a so-called "rod."* In the Heteropoda and Cephalopoda Grenacher* has shown the cuticular rod to be formed of two gutter-shaped half-cylinders. The adjoining half-rods are so disposed that commonly four of them, belonging to as many cells, join to form a single rhabdom. This structure I have found to be easily demonstrated in transverse sections through this region in the cuttlefish. The analogy between the peripheral rhabdom and the true central rhabdom of the arthropodous eye must not, however, be pushed too far; there is no close association of a group of cells here, as in the retinula of insects, &c.

* "Abhandl. zur Vergleich. Anat. des Auges." I. Die Retina der Cephalopoden, 1884. II. Das Auge der Heteropoden, 1886.

IV. *The cuticular ends of the cells in invertebrates are turned towards the light.* To this rule the eyes on the edge of the mantle of Spondylus and Pecten, and on the back of Onchidium, form exceptions. The process of development of these structures has not yet been ascertained, but doubtless there is a process of invagination to account for this inversion. In the case of the Pecten, at any rate, the cuticular ends of the inverted cells may be considered as really turned towards the light, in as far as the eye is lined posteriorly by a tapetum from which the light is reflected, so that the inversion is physiologically an apparent rather than a real one.*

V. *The sight-cells in all eyes on the "camera obscura" type are arranged with their long axes parallel to the incident light, either directly, or as modified by lenticular refraction.* This arrangement is very probably due to the action of phototaxis, whose influence we have already considered in connection with vegetable cells.

VI. *Pigment is universally present, either in the light-percipient cells themselves, or in cells situated in close relation to them.*

In Vertebrates the visual cells are not formed *directly* from the surface ectoderm. By a growth of lateral ridges in the ectoderm or epiblast, which gradually arch over and ultimately unite, a long tube is formed, from the walls of which the central nervous system subsequently arises. In the front part of the embryo this tube is wider than posteriorly, constituting the cerebral vesicle from whose walls the brain and retinæ are differentiated. The ectodermal cells which were superficial now line this primitive nerve tube and its vesicular enlargement, and persist as the cells lining the central canal of the future cord and the central ventricles of the brain. The free cuticular border of

* For this suggestion I am indebted to Bertkau, who gives this explanation of the situation of the rods in the posterior eye of the spider. ("Beiträge zur Kenntniss der Spinnen." Arch. f. Mikr. Anat., Bd. xxvii, p. 589.)

these cells is therefore directed towards the cavity of the central canal and ventricles, and on this border cuticular structures in the form of cilia are often found to be developed.

A somewhat similar though simpler process obtains in the developing Ascidian, and in this animal there is a single special development of the enclosed ectodermal cells on the dorsal wall of the cerebral vesicle, accompanied by a similarly localised formation of pigment around the inner free or cuticular ends of these modified cells. We have here the earliest formation of a retina on the vertebrate type. The visual cells, whose formation we have just indicated, are large and pyramidal in shape, arranged radially, and converge inwards towards the pigment mass. The wall of the cerebral vesicle is bulged outwards in the position of this primitive eye.

In the lowest vertebrate, the *Amphioxus*, the pigmented eye-spot is also single, and is probably similarly developed, but nothing positive has yet been determined on this point; the retinal cells are certainly not more highly differentiated than in some of the *Tunicata*.

Recently, a further proof of the primitive vertebrate eye being a single central pigmented body on the type of that found in the Ascidian and other Tunicata, has been adduced, mainly through the researches of Spencer* and de Graaf† on the pineal body in the *Lacertilia*. This body is part of the *epiphysis*, which consists in a hollow outgrowth of the wall at one part of the cerebral vesicle. The termination of this epiphysis is dilated into a more or less spherical sac, having its walls formed of epithelium; in the Lacertilia we may denominate this the optic vesicle. From the anterior part of the wall of this vesicle the lens is formed, while from the posterior wall come the retinal

* "On the Presence and Structure of the Pineal Eye in Lacertilia." Qu. Journ. Micr. Sc., vol. xxvii. Also, "Parietal Eye of Hatteria." Journ. Roy. Micr. Soc., vol. vi, p. 580.

† "Zur Anat. u. Entwick. der Epiphyse bei Amphibien und Reptilien." Zool. Anzeiger, No. 219, p. 191.

structures. Now it must be recollected that the cuticular ends of the cells lining the vesicles are directed inwards, consequently the rods are formed on the inner or anterior ends of the cells, *i.e.*, facing the light, a direction similar to what we have found to be the rule among the invertebrata. It is not correct, however, to speak of this eye as being developed on the invertebrate type, as has been done; its primitive development is quite on the usual vertebrate lines, and the first divergence occurs when the lens comes to be formed of the cerebral epithelium, instead of by a process of cuticular epiblast. The first *idea*, therefore, is the same for this median, practically extinct eye, as for the persistent paired eyes of vertebrates. In the median eye the primary optic vesicle persists as the eye-cavity, while in the paired eye the primary optic vesicle is obliterated, and the eye-cavity is formed secondarily quite in front of the parts developed from cerebral epithelium.

The true vertebrate retina is in like manner formed from a part of the included cerebral ectoderm. In bony fishes I find that it arises as a *solid* outgrowth from the central nerve-mass, but in cartilaginous fish, and in all other vertebrates, it is a hollow prolongation of the wall of the cerebral vesicle. Subsequent changes, coincident with the development of the lens from the superficial ectoderm, cause the termination of this prolongation or *primary optic vesicle* to be bent inwards, so that we get a cup formed by its two layers, which are thus brought into apposition. From the anterior layer the retina proper is developed, while from the posterior we get the pigment epithelium. It follows from this mode of development that the cuticular surface of the anterior layer is directed backwards, and the rods formed from it are consequently directed *away from* the light.

The retina consists of three great divisions, viz., *Pigment*, *visual epithelium*, and nervous layer or *retinal ganglion*. The visual epithelium represents, as we have seen, the epithelium of the primary optic vesicle, and is homologous

with the epithelium of the central canal of the spinal cord. Each visual cell consists of rod or cone, with its nucleus, fibre, and bulb. To the retinal ganglion belongs the remainder of the retina proper, including thus the two molecular layers, the inner granules, the layer of ganglion cells, and of nerve-fibres. The visual epithelium and retinal ganglion are held in position by a supporting framework of connective tissue. Blood-vessels are present in the retinal ganglion of a large number of vertebrates, and lymphatic spaces have also been described in this position.

I shall now consider more at length the layer of *visual epithelium*, including under this title the rods and cones with their cells, the nuclei of the latter being commonly known as the "outer nuclei" or "outer granules."

The size of the individual cells varies much both in length and breadth in different vertebrates. They may be divided into rod- and cone-cells from the nature of their outer extremities. Each outer extremity is subdivided into an outer and inner segment. Cones are shorter and thicker than rods, especially in amphibians, and they have tapering outer segments, while the outer segments of the rods are straight and cylindrical. Forms, however, sometimes occur which are in some respects like cones, in other respects like rods.

Both outer and inner segments are to be regarded as cuticular structures; the latter, according to Krause, corresponding to the bulbs of cilia. The outer segments of both rods and cones easily break up after death into numerous transverse discs, which possibly correspond to cuticular shedding. The outer segments of the rods have a uniform reddish-purple colour during life, except in some nocturnal animals, as the goat-sucker and the bat, and in the hen and pigeon among diurnal birds. In some amphibians a few rods occur with short *green* outer segments.

The outer segments of the rods and cones exhibit

a difference between their central and cortical substance, recognised by unequal refractive power. There is also a well-defined border, corresponding to an external husk of neuro-keratin. On treatment with weak chloride of sodium solution, the axial part of the rod is dissolved, and the discs fall asunder as rings, so that probably both the axial part and the intermediate substance consist of a form of globulin. The peripheral part, constituting the ring thus formed, behaves like the medullary sheath of nerves, and is called rod-myeloid by Kühne.

The inner segments of both rods and cones are thicker in the middle than at the two extremities. The outer extremity of the inner segment is always different in appearance from the remaining part, having a homogeneous aspect and a higher refractive power. The inner part, on the contrary, is faintly granular, and penetrates the external limiting membrane, there being a hole in this membrane corresponding to the size of the segment. On the outer surface of this segment may be seen a fine longitudinal striation, which, according to Max Schultze, is due to fine fibrils proceeding from the limiting membrane, and seemingly serving for stability. The parts of the rods and cones outside the membrane are surrounded during life by an albuminous fluid, and this striated appearance may possibly be due to its coagulation, or to a shrinking of the outer shell of the segment on its contraction.

In most vertebrates, with the exception of man, the highly-refractive part just mentioned is very distinctly marked off, being ellipsoidal in shape, and named in consequence the rod- or cone-ellipsoid.

The rods are relatively of large size in Amphibians, and even Birds have larger rods than mammals. In osseous Fish, however, they are exceedingly fine, and contrast strongly with the large cones. Among mammals there is considerable variety in the size of the rods, those of the rat being the finest of all.

In Amphibians, Reptiles, and Birds, there is a round fatty globule at the extreme outer end of the inner segment of the cone; it is sometimes colourless, but often coloured red, yellow, or bluish-green. In the sterlet, a ganoid fish, a colourless oil globule also occurs in this position; this was first observed by Bowman, who compared these globules with those of Birds. I have observed similar fatty globules in the cones of all the marsupials examined fresh, with the exception of the American opossum, in which rods only were found, and although the retinæ of the monotremes that I have obtained were unfavourable for exact observation on this point, I was led to believe that they also existed in the *Ornithorhynchus* at least. They are not found in any higher mammals.

Most vertebrates have both rods and cones, the former considerably in excess except in Birds. In the nocturnal birds of prey, however, the cones are comparatively few in number. Many rodents, as the rat, mouse and guinea-pig, have very few cones, and they are entirely absent in the hedgehog, bat, and mole. Many fish also are said to have no cones, *e.g.*, the sharks and rays, but this is open to question. They certainly have only one form of element, but that seems to me to be intermediate between ordinary rods and cones. In some Reptiles and Amphibians, on the other hand, cones alone are present.

In many vertebrates two cones are frequently joined. In osseous fish this is the rule, and I have very rarely found a true simple cone in their retinæ. Each such *double-cone* is usually of large size, and consists of coalesced inner segments, with two distinct outer segments and two cone-fibres. The body of this double-cone gives only a faint indication of its true nature by a furrow traceable down its side. In ganoid and elasmobranch fish no double-cones occur. In Amphibians, Reptiles, and Birds, both simple and double-cones are found, but here the two associated halves exhibit various differences, partly expressed in inequality of size, partly also in a dissimilar

distribution of the lenticular body, and of the oil globule. These are called *twin-cones*, and the two halves are named respectively the *chief* and the *associated* cone. Transitional forms occur between the simple and twin-cone, some having only one nucleus in common, others two; some having one outer segment in common, though possessing two oil globules and two fibres. In teased preparations of the human retina I have occasionlly found somewhat similar structures, viz., large single cones with two fibres; these possibly also sometimes occur in other mammals, but no true twin-cones have ever been found in this group.

In regard to the cone-ellipsoids of Birds, Dobrowolsky makes some interesting observations. The inner surface of the ellipsoid is the more convex, but in this respect they vary according to the colour of the oil globule in the cone. In those with *red* globules the ellipsoid is generally most convex; then come those with *yellow;* and, lastly, the *blue* show usually only a very slight convexity; often indeed a plane or even concave surface. With the greater or less convexity of the ellipsoid and colour of the globule is associated also a difference in the form and kind of cone; those with red globules are the narrowest, those with blue the broadest. Again, the outer segments vary; those with red globules are longest, those with blue shortest,—the length of the inner segment being just the opposite. These variations, if they actually exist, have very probably a relation to the difference in refrangibility of the different light-waves transmitted.

A narrow neck separates the inner segment of the cone from its nucleus, which commonly lies near the external limiting membrane. On the inner side of the nucleus the cone-cell is prolonged as a rather thin, often curved fibre, which terminates at the outer border of the outer molecular layer in a triangular enlargement, its base resting upon this latter layer. Sometimes we can see that this enlargement or cone-fibre-bulb has a hollowed-out base, so that it is more or less stirrup-shaped, the

cavity being filled by a small deposit of protoplasm. The connection between this bulb and one or more processes of the bipolar median cells has been found by Merkel in teased preparations, and was described in sections of the human retina by myself.

The inner segment of the rod ends inwards in a narrow fibre, which sooner or later swells out to enclose a nucleus. Beyond this point the rod-cell commonly presents the appearance of a delicate varicose fibre, terminating in a small rod-bulb near or on the outer molecular layer, where it is also seemingly joined by a process of a bipolar median cell.

The number of nuclei in, and the consequent thickness of, this layer of "outer granules" are in direct relation to the thickness of the inner segments of the elements. Where these are much thinner than the nuclei, the latter must be disposed in several rows, as their size is not usually much smaller in such cases. The layer reaches its greatest development in mammals, while in many Fish, as also in Amphibians and Birds, there are seldom more than two rows, corresponding to a comparatively small number of visual cells.

Salzer* computes that there are from three to four million of cones in the human retina, and, according to Krause,† there are about 400,000 *large*, and about as many more *fine* fibres in the optic nerve, say a million in all, so that if these calculations be correct, each nerve-fibre must be connected with several cones. From the number of outer processes often found coming from the ganglion cells, this preponderance of visual cells may easily be accounted for.

In many Amphibians, club-shaped bodies, first described by Landolt, are found in the layer of outer granules. They rest on the outer molecular layer, and run outwards

* "Ueber die Anzahl der Sehnervenfasern und Retinazapfen am Auge des Menschen." Sitzungber. d. Kaiserl. Acad. d. Wiss., iii, p. 17, 1880.

† Graefe's Archiv, xxvi, 2, p. 102.

between the outer granules, ending in the neighbourhood of the external limiting membrane in one or two swellings. The nature of these is doubtful, but possibly they may merely be torn off rod- or cone-fibres.

The literature of the histology of the visual epithelium is very large and confusing. I am very doubtful as to the benefit of observations on the more minute modifications in the outer and inner segments, unless made in series by the same observer, with due regard to the previous condition of the animal during life and of the preserving reagents employed.

Two of the most recent writers on the anatomy of the retina, Krause* and Borysiekiewicz, deny the connection of the rods and cones with the recognised nervous elements of the retina, and both affirm that they are in direct connection with Müller's fibres. Krause asserts that one radial supporting fibre corresponds to each cone; but this cannot be the case, as the number of Müller's fibres increases as we approach the *ora serrata*, while that of the cones diminishes, and there is no such increase in the number of fibres at the area centralis as his view would necessitate. Borysiekiewicz† even goes so far as to state that in the retina of a tiger that saw well during life, there were *no* rods and cones. Now I can quite understand that none were present when he examined it, but this was doubtless due to post-mortem changes, which very quickly affect this layer. Indeed, in the retinæ obtained from the Zoological Society's Gardens here, I have often observed the rods and cones to be quite broken down, and sometimes almost the entire retina disorganised. Nor can we be surprised at this result when many hours must occasionally elapse before the attendant discovers that an animal is dead, and some time longer before its body is removed and examined. The curves in the

* "Die Retina." Internat. Monatschr. f. Anat. u. Hist., i, p. 245.

† "Stäbchenorgan der Retina." Wien. Med. Presse, 1883, No. 23. Also his work, "Untersuch. über den feineren Bau der Netzhaut, 1887."

retina, too, which this observer describes seriously as "papillæ" are simply due to crumpling of the retina when softened and detached.

The *pigment layer* consists of a single row of prismatic cells, seen to be hexagonal on a surface view, and to be separated from one another by a little clear intercellular substance. The outer border of the cell rests on the hyaline coat of the choroid, and is colourless; near this border the nucleus is placed. The remainder of the body of the cell contains much pigment, both in the form of granules, and of fine needles of pigment called *fuscin*. Where a tapetum lucidum exists no pigment is present in these cells. From the inner or cuticular surface of the cell arise numerous fine processes, which pass inwards for a considerable distance between the end-elements, sometimes stopping at the division between the outer and inner segments, sometimes reaching the external limiting membrane. These may be colourless or contain pigment-needles according to previous conditions as regards light and shade.

Externally each cell has a covering of neuro-keratin around the central protoplasm, which latter contains, besides the pigment, a substance called *myeloidin* by Kühne, very like, if not identical with, the medullary sheath of nerve-fibres. Sometimes, as in the frog, there is also a yellow oil globule. These cells have the function of restoring retinal purple, and this apparently independently of the pigment they contain.

A *yellow spot* is only possessed by man and monkeys, as the diffused pigment to which the colour is due is absent in other animals. A specialised area at or near the centre of the retina is found in all vertebrate classes, but it is not always in the form of a depression or fovea, so that the term area centralis is the more uniformly applicable one.

The true fovea centralis is a pit-like depression in which the retinal ganglion becomes very thin and in which the rods are wanting, while the cones are much elongated

and thinned, especially so in some Fish and in Reptiles. Berger* describes a depression, which he calls the fovea centralis, as constant in occurrence in Fish at the entrance of the vessels of the vitreous. This observation is in support of Hannover's view that the fovea centralis in all animals is a rudiment of the fœtal optic cleft.

In the Hippocampus† there is a slight depression on the inner aspect of the area centralis, but the retina is really thicker here than elsewhere, on account of the great length of the cones and pigment cells.

Among the Reptilia a very well-marked fovea is found in the chamæleon. Hulke thus sums up the peculiarities of this part in the retina of the chamæleon:—They "consist in the attenuation of the optic nerve, ganglionic granular layers and cone-fibre plexus, from its margin towards its centre, in the absence of these layers at the centre, in the great slenderness and length of the cones, in the deflection of the primitive cone-fibres, and of their continuation in the outer and inner granular layers from the centre towards the periphery; and in the maximum development of all the layers, excepting the bacillary, at successively increasing distances from the centre of the fovea in their order of superposition from the outer to the inner surface of the retina."

This description would apply almost equally well for the fovea centralis in man. Here the ganglion retinæ is quite supplanted at the deepest part of the pit. For some distance around it the layer between the outer limiting membrane and the outer molecular layer is very thick, due to the fact that the cone-fibres run obliquely to be connected with the bipolar median cells and through them with the ganglion cells, which are also bipolar in this macular region. On account of the great thickness of the layer of outer granules and ganglion cells, the retina is

* "Beiträge zur Anat. des Sehorgans der Fische." Morphol. Jahrb., Bd. viii, 1, p. 97.

† Carrière. "Die Sehorgane der Thiere," 1885, p. 57 and fig. 39.

considerably thicker around the fovea centralis than anywhere else. At the fovea the retina is not only concave on its vitreous aspect, but also outwards, and the space included between the outer and inner limiting membranes is thus rendered very narrow.

In Birds an area centralis is found at or near the middle of the fundus, and not uncommonly a second area of acute vision, known as the "red field," from the preponderance of red globule-cones, is found in the upper temporal part of the fundus. These latter serve frequently for binocular vision, as the image of an object lying in front and a little below falls at the same time on both red fields. Hence we can understand how birds avoid the extreme convergence that would be necessarily associated with near vision if they only possessed central acute areas; if we watch a hen, for example, picking up its food we recognise the absence of convergence. In the whole binocular visual field of a bird, consequently, there are three areas of distinct vision, viz., two central and one lower nasal. This accounts to some extent for their wariness, and for the side glance they often regard one with. Indeed, we can learn a great deal as to the presence or absence of an area centralis by the manner in which animals move their eyes: if they fix the object regarded, they must have a particular region of more accurate vision.

Besides the red globule-cones present in the red field of the bird, we have also orange and yellowish-green ones in the same area. The red ones are here, however, quite peculiar, in that thay have not only a large red globule in the usual position, but also tiny red drops throughout the protoplasm of their inner segments, extending close up to the outer limiting membrane and principally collected in the outer part, viz., in the opticus ellipsoid.

The changes produced in the vertebrate retina by the action of light may be classified as: (1) *Photo-Mechanical*, (2) *Photo-Chemical*, and (3) *Photo-Electrical*.

(1.) *a.* The best known photo-mechanical change consists in a wandering of pigment inwards within the fine protoplasmic processes of the pigment-cells formerly mentioned. A short exposure to a bright light suffices to bring about a distinct wandering of the pigment, and the more refrangible rays seem to act most powerfully, the red rays least, and the yellow rays with an intermediate strength. There is still some doubt whether this change is purely due to the effect of light on the pigment granules, and is not induced by changes produced in the cell-protoplasm. According to Angelucci,* the processes do not change their position; but I am inclined to think they do, for the following reason. On examining the retina of a frog that had been kept in darkness, the pigment-epithelium was found loosely attached to the retina proper, and there was some difficulty in retaining it in position for examination. In a retina that had been exposed to light during life, on the other hand, the pigment epithelium adhered to the rods and cones, suggesting that their outer segments were in this instance more tightly grasped by the swollen and elongated processes. This protoplasmic movement may, however, be an indirect effect due to primary changes produced in the pigment granules. In favour of this view is an observation of Gradenigo,† that exposure to heat alone will induce the pigmentary changes. We may, therefore, readily understand that the pigment, by changing the light-energy into that of heat, may provoke protoplasmic movement, which will secondarily produce movement of the pigment particles. I find, in the frog, that it is the pigment-needles, not the granules, that wander, and they arrange themselves with their long axes parallel to the length of the processes.

* "Une Nouvelle Théorie sur la Vision" (trad.). Recueil d'Opht., 1885, p. 220.

† "Intorno all' influenza della Luce e del Calore sulla Retina della Rana." Padova. Stabilimento Prosperini, 1885. (See Ref. in Hoffmann's Jahresb. für 1885.)

As Engelmann has pointed out, this movement of the pigment can be produced by illumination of the surface of the body alone. This reminds us of the well-known alterations in the form of the contractile pigment cells of certain fish, amphibians, and reptiles induced by changes in illumination, though the effect is here different. Lister, to whom we are indebted for first directing attention to the contraction of these cells in the frog on exposure to bright light, regards the action as an indirect one, communicated by nerves, principally in the form of a reflex, from the eye. The reflex contraction of these cells may be regarded as similar to the reflex contraction of muscle-cells.

In this connection also may be mentioned the changes produced by light and shade on the arrangement of the chlorophyll in the cells of plants. From the experiments of Sachs, Frank, and quite recently of Moore,* this is due to the action of light on the protoplasm, and, like the changes in the retinal pigment, is induced particularly by rays of high refrangibility.

b. Recent observations of van Genderen Stort,† and Engelmann have made us acquainted with a second example of photo-mechanical action in the vertebrate retina, viz., a shortening of the inner segments of the cones under the action of light, and an elongation in darkness. On exposure to light, therefore, the outer segments are drawn towards the outer limiting membrane and *away from* the pigment cells, while in darkness the opposite movement takes place. This effect has been observed in the retina of all animals hitherto examined, viz., in Fish, Amphibians, Reptiles, Birds, and Mammals, including man. In frogs, fish, and pigeons the movement is particularly easily seen, and the contraction has been observed to be constant even on feeble illumination. The part which

* "The Influence of Light upon Protoplasmic Movement." Spencer le M. Moore, Journ. Linn. Soc., vol. xxiv, p. 200, 1887.

† "Ueber Form- und Ortsveränderungen der Netzhautelemente unter Einfluss von Licht und Dunkel. Graefe's Archiv f. Ophth., Bd. xxxiii, Abth. 3, p. 229, 1887.

moves actively is that which in its optical and chemical properties is like protoplasm, viz., the part of the cone between the ellipsoid and the cone-nucleus, and to this they give the name of "cone-myoid." I would suggest that the peculiar form of the outer segments of the cones is associated with this action, in being such as will favour a rapid elongation into the pigment-cell in darkness.

All parts of the visible spectrum, on sufficient duration of exposure and strength of the illumination, can excite this reaction of the cones, as also of the pigment; like the pigment-cells, however, the cones also react most strongly to the more refrangible rays.

In the Bird, all cones are contractile, irrespective of the colour of the oil globule they contain. Since the same rays which are absorbed by the coloured globules act strongly in cones in which the globules lie at the junction of the outer and inner segments, but are nearly powerless in cones where they lie scattered throughout the inner segment (as in some of the elements of the so-called "red-field" of the bird's retina), it follows consequently that the locality of the primary irritation lies in the inner segment itself.* In the case of all coloured globule-cones, however, I would remark that the pigment-cells will only be exposed to the transmitted rays, and I would suggest that in this way they will have an influence upon the total light-effect produced, as I shall endeavour to show later.

(2.) *a.* Photo-chemical action is illustrated by the bleaching of the retinal purple in the rod outer segments on exposure to light,—and by its re-secretion in the pigment-cells, especially in darkness.

In the case of some fish, *e.g.*, the Bream, this process can be observed during life, from the fact that the fundus

* Engelmann en van Genderen Stort. "Nieuwe mitkomsten betreffende de be bewegingen van kegels en pigment in de retina onder den inloed van het licht." Onderz. in het. physiol. Labor. te Utrecht, ix, p. 145 (Ref. in Virchow and Hirsch's Jahresb. f. 1884).

is white, due to the presence in the pigment epithelium of an opaque, chalky material called guanin.* Here it is bleached very gradually during life, taking at least 20 minutes of exposure to direct sunlight, while it is regenerated after about an equal interval of time.

During the bleaching process the visual purple is first converted into a substance named by Kühne "visual-yellow," which in turn is further changed into "visual-white."

All visible light decomposes it, but, intensity remaining the same, at very different rates. "The entire beam of white light is the best transformer of visual purple—better than light of any particular wave-length." It is seemingly only acted on by the visible spectrum, and "of all the visible rays, those bleach the visual purple most freely and quickly which the visual purple in solution most effectually quenches. Thus the order of activity is yellowish-green, green, blue, greenish-yellow, yellow, violet, orange, and red."† "Rays of such wave-lengths as rapidly convert *visual purple* into *visual yellow*, act most slowly on the latter: those which most easily change the *visual-yellow* into *visual-white*, and which are most readily absorbed by the *visual-yellow*, have, as a rule, a weaker action on the visual purple itself."‡

It is of course not essential to vision, as it does not occur in the cones, and the more recent researches on this subject have rendered exceedingly doubtful its having any real connection with either ordinary vision or colour perception. I would suggest that its function is probably to protect the pigment epithelium from too rapid exhaustion where great activity is unnecessary, *i.e.*, away from the visual axis.

It is important to remark that it is absent or feebly

* Kühne and Sewall. "On the Physiology of the Retinal Epithelium." Journ. Physiol., iii, p. 88.

† Gamgee. "Physiological Chemistry."

‡ Kühne on Retinal Purple. Transl. by Michael Foster.

developed in birds where coloured oil-globules exist in the cones, and that these globules are also bleached by exposure to light. They possibly serve in a similar manner to protect the pigment-cells from too great an action of certain rays, viz., of those absorbed by the oil-globule in question.

b. Another photo-chemical effect on the retina that is as yet very imperfectly known, will I think ultimately prove to be of much importance, viz., the alteration in the reaction of the contents of the pigment-cells due to light-exposure. This seems to be an oxidising process, accompanied by the substitution of an acid for a previously alkaline reaction.

(3.) From the investigations of Holmgren, Dewar and McKendrick,* and more recently of Kühne and Steiner† and of Chatin,‡ it has been conclusively demonstrated that the action of light upon the retina is accompanied by an increase of the normal electrical current passing inwards along the optic nerve to the brain.

This action was found by Dewar and McKendrick in many vertebrates, high and low, and also in Crustacea; Chatin has in addition proved its existence in Molluscs.

Different parts of the spectrum produce different amounts of increase, the yellow rays having the greatest power. From this part of the spectrum we have a gradually diminishing action on either side until we reach the ultra-red rays on the one hand, or the ultra-violet on the other, when it is found that no change whatever is produced in the electrical current by these non-luminous waves.

Cohn, again, in his experiments on the furthest limits

* Rep. of Brit. Assoc. for Advancement of Science, 1875.

† "Ueber Elektrische Vorgänge im Sehorgane." Unters. aus Chem. Phys. Inst. zu Heidelberg, iv, p. 64, 1881.

‡ "Sur la valeur des impressions monochromatiques chez les invertébrés." Compt. rend., t. xc, p. 41. Also, "Action des lumières colorées sur l'appareil optique des Crustacés." Gaz. Méd. de Paris, 1880, p. 363; and "Contrib. à l'étude des phenomènes de la vision chez les gastéropodes pulmonés." *Ibid.*, p. 56.

of colour-perception, finds that a yellow spot is seen to be bright at the greatest distance: the order of greatest distance of visibility in sunlight is 1, yellow; 2, orange; 3, green; then 4, red, blue, and violet with the same greatest distance. Nettleship, from his investigations conducted in daylight, came to very similar conclusions. Now this order of visibility is exactly what we would expect from the action of those different wave-lengths in influencing the electrical current: the middle of the spectrum is most powerful, and the extremities are least so.

It would appear, therefore, on comparing these series of investigations, as if the amount of the afferent electrical current were coincident with changes in the visual centre associated in our consciousness with impressions of colour.

From all the foregoing known facts regarding light-percipient organs, I would draw the following main general conclusions:—

1. That all light-percipient cells are modifications of epithelial cells, or developed from the same embryonic layer which forms them.

2. That the ends of these cells corresponding to the cuticle have generally cuticular structures formed in the shape of rods.

3. That the opposite ends of these cells are either directly continued as nerve-fibres, or are connected with ganglion cells, and ultimately with nerve-fibres.

4. That pigment is practically always present, either in the light-percipient cells or in close connection with them. Exceptions exist where a tapetum is present, and in the rare albino individuals having eyes of high types, but then non-pigmented cells always exist, which are evidently capable of performing similar functions to those of the pigmented cells, though in a minor degree.

5. That all forms of vegetable and animal protoplasm sensitive to light are acted on by the shorter waves corre-

sponding to the violet end of the spectrum, and that the same holds good in a marked degree in the case of all those light-percipient cells as yet known to act to light.

6. That the property of *phototaxis* is universally influential in the manner in which these cells arrange themselves, and in the direction in which they contract on exposure, *i.e.*, parallel to the incident light in both cases. When the cell is free, as in the Euglena and Zoospore, this effect exhibits itself in free phototactic motion, while in the case of attached retinal cells it shows itself in contraction towards the incident light, *i.e.*, in positive phototactic movement. The Euglena may thus be looked upon as somewhat similar to a red globule-cone in birds as regards its action to light.

7. That the pigment-cells have evidently the property of secreting chemical fluids according to their exposure to light and shade, and that the pigment contained in them is doubtless of service as being light-absorbent, and thus aiding a rapid transformation of light-energy into protoplasmic action.

8. That the result of the action of the secretion of the pigment-cells on the segments of the light-percipient cells may be demonstrated by the electrical current produced.

9. That seemingly the strength of this current depends upon two factors, viz. :—1, the activity developed in the pigment-cell under the action of light, and 2, the depth to which the cone is embedded in it, *i.e.*, the extent of surface exposed to its action.

10. That in the case of vertebrates at least, this resultant current is greatest on exposure to yellow light, *i.e.*, to visible waves of medium length and refrangibility.*

11. That differences in the action thus produced, and

* Thus in the case of exposure to violet rays the action of the pigment-cell is greatest, but the contraction of the cone is also greatest, so that a relatively small portion of it is exposed to the action of the pigment-cell. In the case of red light the contraction of the cone-myoid is least, but the action in the pigment-cell is also least, so that although a large area of the cone is embedded in the pigment-cell the total effect is relatively small. In the case

consequently in the nerve-current transmitted to the visual centre, are associated in our minds with sensations of differences in colour.

12. That as regards the stationary rods, the action induced will probably depend upon the amount of destruction of retinal purple, and the consequent demand upon the pigment-cell. As this action is known to depend more on intensity or wave-height than on wave-length, it is improbable that the rods have any higher function than that of transmitting impressions which will be appreciated in our conscious centres as variations in light-intensity.

13. The function of the retinal purple is probably mainly to protect the pigment epithelium from too great exposure, and thus to modify its secretion and its action on the end-organs embedded in it. Were the peripheral area of the retina not defended in this manner, strong lateral illumination would have an unnecessarily great and confusing effect. It must be borne in mind that the retinal purple is constantly being regenerated in the living retina during exposure to *diffused* light at any rate. The result of its presence may therefore be compared with that of tinted glasses, viz., preventing too great a light-action on the pigment-cells.

of yellow light, the action on the pigment-cell is considerable, and the contraction of the cone-myoid not sufficient to prevent much of the cone being embedded in the pigment and acted on by it, so that the total resultant effect is great.

CONCOMITANT CONVERGENT STRABISMUS.

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This communication must be read only in conjunction with our previous article on this subject in the last Part of these Reports, since the preliminary steps in our investigation are there described.

In this essay we have tabulated a number of facts obtained from methodic clinical investigation, and have formulated some principles which may be deduced from those facts.

We may say that in every case in which a statement of the refraction is made it represents the results obtained by retinoscopy under atropine or homatropine and cocaine. Subjective methods throughout have been discarded as much as possible.

We regard strabismus cases as alternating only when the patient squints with each eye indifferently. Those cases in which, notwithstanding excellent vision and power of fixation, the patient squints with one eye habitually, we exclude from the category of alternating cases.

I. *Possibility of Alteration of Vision in the Squinting Eye.*—We obtained the distant vision in the squinting eye after accurately correcting the refraction. The fully correcting glasses were then prescribed, and were worn by the patient for months or years. The distant vision was then again tested. The following table gives the results obtained:—

No.	Changes in distant vision.	Duration of treatment.	State of strabismus.	Vision (uniocular or binocular).	Remarks.
1	From $\frac{6}{36}$ to $\frac{6}{24}$.	7 months	Cured 3° in.	Uniocular.	
2	Nil	3 months	Improved from 40° in to 12° in.	Uniocular.	
3	From < $\frac{6}{60}$ to $\frac{6}{36}$.	9 months	Improved from 35° in. to 25 in.	Uniocular	The other eye was covered and eserine was frequently applied to the squinting eye for some months.
4	From $\frac{6}{12}$ to $\frac{6}{6}$.	2 years	Improved (alternate strabismus).	Uniocular.	
5	Nil	6 months	Cured 0°	Binocular.	
6	Nil	12 months	Improved from 28° to 13°.	Uniocular	The healthy eye was covered up several times, but the patient fell over chairs, &c.; was quite unable to see.
7	Nil	6 months	Cured 0°.		
8	Nil	5 years	Cured 0°.		
9	Nil	9 months	Improved by glasses and tenotomy from 35° to 25° to 10°.		
10	Nil	12 months	Cured 0°	Uniocular.	
11	Nil	14 months	Cured 5°.		
12	Nil	16 months	Cured 0°	Binocular.	
13	Nil	12 months	Cured 0°	Uniocular.	
14	Nil	4 months	Cured 0°	Uniocular.	

We have included in the foregoing table one case of alternating strabismus (4) in which the vision in one eye was less than in the other.

Of the 14 cases recorded, in 11 the constant use of glasses failed to effect any improvement whatsoever in periods varying from 3 months to 5 years, although in these 11 cases the squint was cured in 8 cases and improved in the other 3, and also although the resulting vision was binocular in 2 of the cured cases.

In the other 3 cases, including the alternating cases, some improvement in distant vision resulted.

Thus glasses which produced great improvement in vision in the other eye, and even re-established binocular vision, absolutely failed to improve the vision in the amblyopic eye. Therefore this evidence tends to show that in cases of concomitant convergent strabismus treatment is of value chiefly for cosmetic purposes.

In so far as we are aware there is no definite evidence to show that valuable improvement in the vision of the amblyopic eye ever takes place under any circumstances. At all events, as just demonstrated, the means we adopted totally failed to produce it.

It seemed to us, however, that much of the alleged faulty vision in such eyes might be due to errors of refraction, particularly as the squinting eye is so frequently astigmatic (*vide* "Refraction in (Concomitant) Convergent Strabismus," p. 10). We accordingly obtained the distant vision in those eyes, then worked out the refraction by retinoscopy under atropine or homatropine and cocaine, and retested the vision with the correcting glasses, with the following result:—

II. Average distant vision in 78 amblyopic eyes in cases of concomitant convergent strabismus without glasses = between $\frac{6}{60}$ and $\frac{6}{36}$.

Average vision in the same 78 eyes after full correction in the manner indicated = between $\frac{6}{36}$ and $\frac{6}{24}$.

In 41 cases, however, the correction did not produce any appreciable alteration, so that in the remaining 37 cases the improvement in vision effected by the glasses was about two points, from $\frac{6}{60}$ to $\frac{6}{24}$.

In 19 cases the vision was less than $\frac{6}{60}$ after full correction.

In 3 cases before correction the vision was more than $\frac{6}{12}$.

In 8 cases the vision after correction was more than $\frac{6}{12}$ — $\frac{6}{12}$ in one case, $\frac{6}{9}$ in two, and $\frac{6}{6}$ in five.

In the light of subsequent investigation it became interesting to see whether there was any special cause for the retention of such excellent vision in these cases. Inquiry showed that in 6 no special cause could be assigned, but that in 2 the squint appeared late in life—in one at six and a half, and in the other at nine years of age. These cases cannot be regarded as cases of alternating strabismus, because, although the power of fixation was excellent, the individual habitually squinted with the one eye only.

The varying amount of vision in these 8 cases before and after correction with glasses reminds one that the use of the term amblyopia must be quite relative. The vision may be $\frac{6}{9}$ and the eye yet be amblyopic.

In these 8 cases—

	Vision before correction.	Vision after correction.
1	$\frac{6}{6}$	$\frac{6}{6}$
2	$\frac{6}{6}$	$\frac{6}{6}$
3	$\frac{6}{36}$	$\frac{6}{6}$
4	$\frac{6}{6}$	$\frac{6}{6}$
5	$\frac{6}{24}$	$\frac{6}{9}$
6	$\frac{6}{18}$	$\frac{6}{6}$
7	$\frac{6}{12}$	$\frac{6}{9}$
8	$\frac{6}{60}$	$\frac{6}{12}$

We endeavoured to find out whether squint is to any extent hereditary, but prior to referring to this point we must state our experience relative to its existence in more than one member of a family.

III. Of 87 cases in which we were able to make careful inquiry for the existence of strabismus in the brothers or sisters of the squinting patient.

In 62 the patient appeared to be the only member affected, the average number of persons in each family being 5.

In 25 cases more than one member was affected. In these cases the average number of persons in each family was 6. 53 individuals squinted, or rather more than 2 per family.

Thus in the great majority of cases, but a small percentage of persons squint in any one family; exceptional cases we have met with where 2 members in a family of 2, or 3 members in a family of 5, have been affected.

IV. We carefully inquired as to the existence of strabismus in the parents or parents' families in 77 cases with the following result:—

In 55 (71 per cent.) of the 77 cases a negative reply was returned.

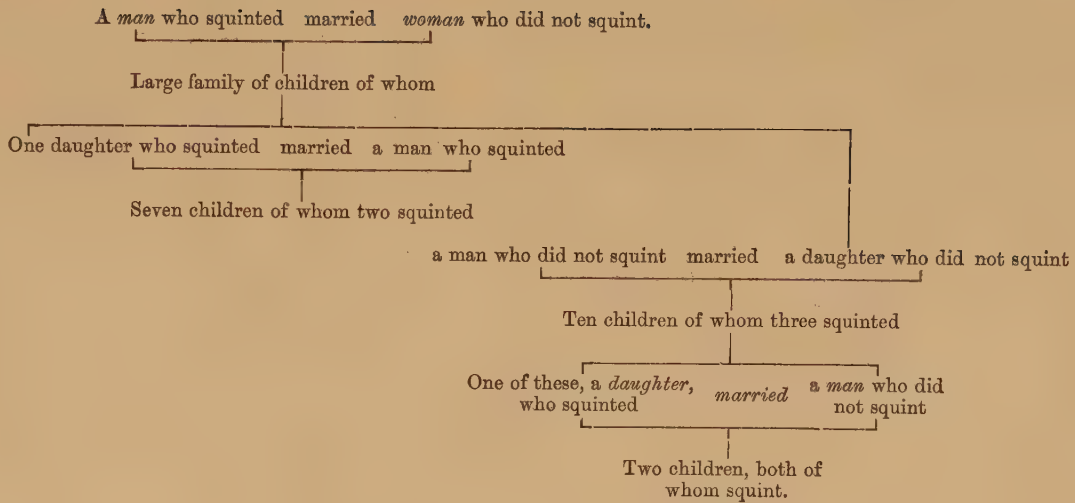
In 22 (29 per cent.) a definite affirmative reply was obtained.

The details of these 22 cases were as follow. In 8 the parents themselves squinted; in 3 the father alone; in 2 the mother alone; in 3 the father, and 2 members of his family.

In the remaining 14 cases, other relatives than the parents squinted: viz., in case (1) the grandmother; in case (2) an uncle; in case (3) a paternal uncle; in case (4) 16 members of the father's family stated to have squinted, but the father himself did not; in case (5) 1 member of father's family (8 in number); in case (6) 2 members of father's family (9 in number); in case (7) 1 member of father's family (6 in number); in case (8) 2 maternal aunts (family 8 in number); in case (9) 1 member of father's family (7 in number); in case (10) 2 maternal uncles; in case (11) 2 maternal uncles; in case (12) paternal aunt; in case (13) maternal cousin; in case (14) maternal uncle and grandfather.

In these cases, no member of the family squinted other than those specified, so that, although in most cases hereditary transmission can be negatived in a fair number of cases, the evidence seems fairly conclusive as to transmission.

But the following case furnishes absolutely convincing evidence of the possibility of transmission.


A *man* who squinted married *woman* who did not squint.
Large family of children of whom
One daughter who squinted married a man who squinted
Seven children of whom two squinted
a man who did not squint married a daughter who did not squint
Ten children of whom three squinted
One of these, a *daughter*, who squinted
married
a *man* who did not squint
Two children, both of whom squint.

The singular interest of this case lies in the fact that two squinting people marrying, did not produce a large percentage of squinting children. In other words, it seems as if it is not the squint which is transmitted, but the liability to develop it: that is to say, it requires a special conformation and special exciting circumstances to develop it.

We endeavoured to find whether amongst the families of those who squint, we could find persons possessing amblyopic eyes, but who did not and never had squinted. In fact, we attacked the question, Is squint a result of congenital amblyopia?

V. In 22 families in which we personally examined the vision and eyes of parents, and in which at least one child squinted, we found two in which one parent was amblyopic. In one case the mother was amblyopic, and two of her children squinted (case C). In the other case the father was amblyopic. Then we examined in the same way the members of 23 families each of which contained at least one squinting individual. In most cases we examined all the members, and estimated their refraction if necessary.

Of these, in 20 families no amblyopia existed. In two cases, just referred to, there was parental amblyopia. In one case a brother was amblyopic.

These figures therefore enable us to state that this congenital amblyopia is not frequently present in the families of those who squint.

VI. We endeavoured to ascertain the age at which the patients began to squint and obtained the following results:—

In 195 cases (including alternating strabismus), the average age at which it began was 3·4 years.

In 36 cases it appeared during the first year of life. In 34 cases it appeared later than the fifth year, and in 3 cases beyond the 10th year. Of these 34 cases, no less than 10 were cases of alternating strabismus in which the

vision in each eye was excellent, and 5 were produced by corneal ulcers or inflammation. Of the remaining 19 the vision taken in 17 after correction with glasses averaged $\frac{6}{18}$, in 4 cases being $= \frac{6}{9}$ or more.

These facts are of great importance; from them it is obvious—

(1.) That good vision in the squinting eye is retained usually in cases of strabismus in which the disease appears later in life than usual (*i.e.*, after 5 years of age).

(2.) That many of the cases which begin late in life are cases of alternating strabismus in which excellent vision exists in each eye.

(3.) That in a fair number of these cases of late origin, the extinction of vision by nebulæ seems to cause the strabismus to appear.

VII. The causes for strabismus assigned by the patients or friends were as follow:—

Pertussis, 23.
Teething, 19.
Measles, 16.
Falls, 8.
Fits, 5.
Fright, 4.
Bronchitis, 2.
Hemiplegia (infantile paralysis?) 2.
Scarlatina, 1.
Inflammation of the eye, 1.
Inhalation of chloroform, 1.
Typhoid fever, 1.
Rötheln, 1.
Nil, 27.

Total, 111 cases.

The usual statement was that the child was attacked by one of these diseases, and that during convalescence they observed the strabismus for the first time.

Special Cases of Strabismus.

(A.) T. L., æt. 8. Right convergent strabismus which was first noticed at 6 years of age. R. V. $= \frac{6}{6}$ Hm. 2 D. L. V. $= \frac{6}{6}$ Hm. 2 D. Here no deterioration of vision has occurred.

(B.) S., æt. 17. Right convergent strabismus which was first noticed at 3 years of age. Has never worn spectacles. R. V. $= \frac{6}{36}$ c. sph. + 2·5 D.Cyl. − 4 D. V. $= \frac{6}{6}$. L. V. $\frac{6}{9}$ Hm. 2·5 D. $= \frac{6}{6}$.

Here the squinting eye has been functionally amblyopic for 14 years. Why has no deterioration of vision occurred?

(C.) Case in which the mother was amblyopic and two of her children squinted (6 in family). Mother æt. 35. R. V. $= \frac{6}{24}$ c. sph. + 2·5 D.Cyl. + 0·5 D. V. $= \frac{6}{24}$. L. $\frac{6}{6}$ Hm. 1·5 D.

Father æt. 39. R. V. $= \frac{6}{6}$ Hm. 0·75 D. L. V. $= \frac{6}{6}$ Hm. 0·75 D.

One child æt. 10. Right convergent strabismus. R. V. $= \frac{6}{60}$ c. sph. + 2·5 D.Cyl. + 3 D. $\frac{6}{36}$ L. V. Hm. 1 D. $= \frac{6}{9}$.

Another child æt. 4. Right convergent strabismus. R. V. $= \frac{6}{36}$. L. V. $= \frac{6}{9}$. By retinoscopy both eyes were hyperopic and astigmatic, but could not be worked out.

This case is extremely suggestive. The transmission of errors of refraction and amblyopia on the right side being especially worthy of note.

Our next endeavour was to obtain information relative to strabismus caused by or co-existent with corneal nebulæ.

Cases of Concomitant Convergent Strabismus in which Central Corneal Nebulæ existed in the Squinting Eye.

No.	Nature of squint.	Vision and refraction.	Amount of strabismus.	Assigned cause.	Effect of treatment.
1	Convergent right.	R. H. 3·5 D. L. H. 3·5 D.	20° in.	Central ulcer at four years of age.	In eight months' constant use of glasses reduced to 8° in.
2	Convergent left.	R. V. $= \frac{6}{6}$ Hm. 2 D. L. V. $= < \frac{6}{60}$.		Ophthalmia in childhood.	Cured by tenotomy of left internal rectus.
3	Convergent right.	R. V. $= < \frac{6}{60}$. L. V. $= \frac{6}{6}$.	20° in.	Ophthalmia in childhood.	Glasses failed to improve. Tenotomy of right internal rectus reduced to 7°.
4	Convergent right.	R. V. $= \frac{6}{60}$ Hm. 2·5 D. $= \frac{6}{60}$. L. V. $= \frac{6}{18}$ Hm. 2·5 D. $= \frac{6}{12}$.	50° in.		Glasses reduced to 5° when worn, 12° when not worn, in 12 months.
5	Convergent left.	R. $\frac{\text{sph. } + 4}{\text{cyl. } + 2 \text{ D.}}$ L. sph. + 4 D.	10° in.	Ophthalmia after measles.	Reduced to 8° by glasses in 12 months.

No.	Nature of squint.	Vision and refraction.	Amount of strabismus.	Assigned cause.	Effect of treatment.
6	Convergent left.	R. + 7·5 D. L. + 10.	20° in.		In five months reduced by glasses to 0°.
7	Convergent left.	R. V. = $\frac{6}{12}$ Hm. 0·5 D. $\frac{6}{6}$. L. V. = $< \frac{6}{60}$ − 5 D. = $< \frac{6}{60}$.	10° in.	Ophthalmia after measles at four years of age.	In 12 months reduced by glasses to 5°.
8	Convergent right.	R. V. = $< \frac{6}{60} \frac{\text{sph. + 2·5 D.}}{\text{cyl. + 2 D.}} = < \frac{6}{60}$. L. V. = $\frac{6}{60} \frac{\text{sph. + 4 D.}}{\text{cyl. + 2 D.}} = \frac{6}{9}$.		Ophthalmia after measles at four years of age.	
9	Convergent left.	R. V. = $\frac{6}{6}$ Hm. 5 D. L. V. = $< \frac{6}{60}$ not improved.	30°.	Phlyctenules.	Reduced by glasses to 18° in one day.
10	Convergent left.	R. $\frac{\text{sph. + 2 D.}}{\text{cyl. + 1·5 D.}}$ L. $\frac{\text{sph. + 3 D.}}{\text{cyl. + 3 D.}}$		Ulcers.	
11	Convergent left.	R. V. = $\frac{6}{6}$ Hm. 4·5 D. L. V. = $< \frac{6}{60}$ Hm. 4 D. = $\frac{6}{60}$.		Interstitial keratitis.	

The point worthy of note in connection with these cases is simply that binocular vision without squint in highly hyperopic eyes was presumably procured by an effort until disease blotted out vision in one eye. Convergent strabismus immediately occurred.

It will be observed that these cases were just as amenable to treatment as ordinary cases, perhaps more so.

The Causation of Strabismus.

The first question which requires answering is the vexed one, Is the amblyopia the consequence or the cause of the strabismus? The facts as we have elicited them are mostly these:—In the great majority of cases of concomitant strabismus (excluding alternating cases) the amblyopia is irremediable. Correction with glasses, prolonged monocular use of the amblyopic eye, cure of the squint and even the re-establishment of binocular vision, all fail to produce any valuable improvement in vision, often to produce any improvement.

In a considerable number of cases other members of the family of the squinting patient, or of the parents' families, are also affected with strabismus.

In very few cases, however, do members of the patients' or the parents' families possess non-squinting amblyopic eyes. An examination of the list of cases of strabismus which began after five years of age shows that it contains:—

(1.) A large proportion of cases of alternating strabismus in which the vision in each eye is excellent.

(2.) Cases in which the vision in the squinting eye had been impaired by corneal nebulæ.

(3.) Ordinary cases in which the distant vision in the amblyopic eye was much better than it usually is in the other cases.

These facts suggest strongly that it is only at a very early period of life that the amblyopia is capable of being

produced. That is to say, we must regard the amblyopia as being due not to a deterioration of vision but to a failure of development.

It becomes a question whether such a thing as deterioration of vision after its acquirement ever occurs; attempts to find such cases seem to have failed. There seems to us to be no reason why vision should deteriorate once the visual sense is educated. But it is probable that in these amblyopic cases there is failure to educate the visual sense at the only time in life when the education is possible, viz., when the sense in the other eye is being acquired.

Against this theory of the causation of the amblyopia such cases as Case B may be referred to in which functional amblyopia had existed in a squinting eye since three years of age, without real amblyopia. Practical suppression for fourteen years, beginning at such an early age had not prevented visual education taking place.

Is the Amblyopia ever the Cause of the Strabismus?

If this were the common mode of origin we should expect to find many more amblyopic people, *i.e.*, people who had escaped squinting, and we should certainly expect to find such people in the families of those who squinted.

But although we found many persons who squinted in such families, in only three out of 22 families was any amblyopia discoverable.

In one case (C) the evidence is exceedingly strong, an amblyopic mother had two children who squinted. Such a case is strongly suggestive of this mode of production of strabismus, but the case is isolated.

The notes of the cases of nebulæ show with what readiness squint may be produced if the vision is destroyed or materially impaired, provided that the eyes be sufficiently hyperopic, and there is no doubt that this theory would render many circumstances much more intelligible. Yet in the great majority of cases one is forced

to believe that the amblyopia is produced as a consequence of the squint. A minority of cases of amblyopia are probably congenital in origin, and the amblyopia in these cases probably determines the development of the strabismus.

It seems that the existence of hyperopia is almost essential for the development of squint; and that astigmatism, or irregularities of refraction, frequently exist in the squinting eye. These latter conditions are found so early in life that the explanation of their presence on the ground of arrested development from disuse must be rejected, and one must believe that in many cases they are causally related to the strabismus.

It further seems probable that diseases such as pertussis may determine the development of strabismus. Yet after the enumeration of every possible cause there remains something which cannot be explained—a "further something" which determines the presence of strabismus in one case and the absence of which negatives the presence of the strabismus in an exactly similar case in all other respects. That this something is transmissible seems probable from the records of our cases. We hope to say more of this in a later communication.

The Influence of Atropine on Concomitant Convergent Strabismus.

There has existed apparently a belief amongst ophthalmologists that the instillation of atropine in cases of concomitant convergent strabismus causes diminution in the amount of convergence. We had ourselves noted a fair number of cases in which it either produced the reverse, or no effect at all, and we therefore investigated the matter.

We measured the angle of convergence in a number of cases of strabismus, then administered atropine to both eyes for several days until complete mydriasis and paresis of accommodation was produced and then remeasured the angle. The following result was obtained. Of 38 such

cases the angle remained unaltered in 11, was increased in 11, and diminished in 16. Thus the strabismus was improved in only 16, or 42 per cent., and was actually worse in 11, or 29 per cent. In these 11 cases the average deterioration was 9°, the maximum being 20°. In the 16 cases the average improvement was 16°, the maximum being 30°.

Of 6 cases of alternating strabismus, in 2 no alteration took place, in 2 there was improvement, and in 2 deterioration.

We next endeavoured to ascertain whether the effect produced by atropine is of any service in prognosticating the effect of the constant use of glasses during periods of 3 to 18 months. Of 4 cases in which atropine increased the strabismus, in 3 the use of glasses failed to produce any improvement. In the fourth case the angle was reduced from 30° to 8° whilst the glasses were worn, and to 15° when they were removed. Of 6 cases in which atropine produced no alteration the use of glasses effected material improvement in only 3. In these the average improvement was 15° whilst the glasses were worn and 12° when they were removed.

Of 5 cases in which atropine improved the strabismus the use of glasses produced material improvement in every case. The average improvement was 16° whilst the glasses were worn and 16° when they were removed.

Thus it seems that the use of atropine in a fair number of cases produces a considerable diminution in the angle of convergence. In the majority of cases, however, it fails to do so and in some cases produces an increase in the angle.

It is also apparent that if the use of atropine effects much temporary improvement, the use of glasses will produce permanent improvement. On the other hand, if atropine fails to improve, one cannot say that the use of glasses will be ineffectual as a mode of treatment, but in such cases improvement does not follow so frequently and is not so great as in the other cases.

TWO CASES OF TUBERCULAR DISEASE OF THE EYE.

By J. B. Lawford,
Late Curator of Museum.

Case 1.—Daisy J., æt. 12 months, was admitted to the Royal London Ophthalmic Hospital under Mr. Couper's care, on May 11, 1885, and underwent enucleation of the right eye on the following day.

Family History.—Father and other members of his family are all healthy. Mother very delicate, suffers from winter cough. A maternal aunt died of phthisis.

The patient is the first child; there have been no miscarriages. She has been a weakly child, and the father calls her "ricketty;" has been bottle-fed since she was a month old. She has had no acute or serious illness.

Three weeks before the date of admission, the parents noted that the child's right eye was bloodshot, but thought nothing of it for about a week, when the eyelids began to swell and the cornea looked a little hazy. The child did not seem to be in pain. There had been, so far as was known, no injury to the eye.

Unfortunately no record was kept of the appearances of the eye at the date of operation.

Examination of the Eyeball after Removal.—Globe partially collapsed, the sclerotic having been cut during the operation. At the upper part, closely attached to the outer surface of the sclerotic, is a dirty grey ragged mass, which overlaps the upper margin of the cornea. The tissues about the insertion of the superior oblique and superior rectus muscles are matted by inflammatory exudation. On section of the eyeball, it is evident that the mass at the upper part is directly continuous through a breach in the sclerotic with the material in the interior of the eye. The gap in the sclera is 6 mm. from the corneal margin.

The cornea is clear, the iris close against its posterior surface. The lens, which is clear, is displaced forwards, pushing the iris in front of it.

The entire cavity of the eye posteriorly to the lens is occupied by a soft spongy material, of a muddy grey colour and varying in consistence, very soft in its central part, but denser towards the periphery. It forms a bed in which the crystalline lens lies. To the naked eye no trace of the retina can be distinguished, but a layer of about three times the normal thickness of the choroid lines the sclerotic, and is in the greater part of its extent fairly well differentiated from the softer material internal to it. Close to the optic nerve, however, this layer is continuous with the central mass; and at the upper part passes through the opening in the sclerotic to its outer surface. This layer, which is evidently the altered choroid, is yellowish in colour, and apparently contains very little pigment.

The iris is but little altered, being perhaps slightly thicker than normal. The ciliary body, on the other hand, is much changed, and the ciliary processes only here and there recognisable; at the upper part they are directly continuous with the mass behind the lens.

After hardening in weak and subsequently strong spirit the eye was examined microscopically, sections being made of several different parts. As a result of this examination, I came to the conclusion that the changes were of tubercular character. The choroid is everywhere greatly thickened and infiltrated with small cells; it is almost entirely devoid of pigment; in some places anteriorly, portions of the hexagonal pigment epithelium remain adherent to its inner surface, but in general this layer is entirely destroyed.

Close to the ora serrata, portions of the retina are distinguishable; elsewhere this tunic has entirely disappeared. The portions referred to are greatly infiltrated with small cells, and the vessels are dilated and full of blood. This part of the retina is very closely adherent to the mass in the vitreous chamber.

This mass which represents vitreous and retina contains in its peripheral part large numbers of small round cells, many of which are possibly the remains of the retinal granules; in its central part there are fewer cells, the structure being indistinctly fibrous.

The iris is thickened and infiltrated, and firmly united to the

anterior lens capsule by a thin layer of new tissue full of lymphoid cells. The cornea shows no changes. The portion of optic nerve removed is inflamed; close to its temporal side, and outside its sheath, is a little nodule composed of small cells and connective tissue. The optic nerve sheath is not dilated.

Throughout all the affected tissues of the eyeball there are numerous very typical giant cells, from many of which processes can be traced, and round which a fine network containing small cells exists. There are also many areas of degeneration in which nothing but a granular structure can be made out, and some of which are scarcely at all coloured by the stains employed, while others stain darkly but diffusely. These appearances are so characteristic, that I felt sanguine that I should be able to detect the *Bacillus tuberculosis* in the tissues, either in the eyeball or in the mass attached to the sclerotic above. In my notes of the case made in 1885, I find a statement that bacilli were detected in the grey mass from the interior of the eyeball, and I remember being satisfied in my mind that I had obtained this additional proof of the nature of the disease. However, being unable to find the slides I then made, I have again at this date (June, 1888) examined portions of the choroid, the vitreous, and the nodule on the outer surface of the sclerotic for Koch's bacillus, with an entirely negative result. Mr. E. T. Collins, the present Curator of the Museum, has also kindly stained and examined several sections for me, but could find no bacilli. Thus it seems doubtful if my previous observation was correct. The specimen has been in alcohol during the interval, and the other microscopic appearances are as characteristic now as they were in 1885.

The after-history of this case is important, and I have been able to hear of the child's condition at intervals since the operation. Her health improved decidedly soon after the removal of the eye, and the child has been since, and is now (June, 1888, three years after the operation), in perfect health.

The mother died of acute phthisis in October, 1885.

Case 2.—Rosa W., æt. 8 months, was admitted to the Royal London Ophthalmic Hospital, under Mr. Gunn's care, on

March 2, 1887, and an iridectomy was performed upon the right eye, for the purpose of removing two small nodules, thought to be tubercular. These nodules were about the size of a pin's head, one rather larger than the other. Microscopic examination of the larger nodule stained and teased, revealed a small celled growth with occasional fine strands of connective tissue. No giant cells were found.

No positive opinion as to the nature of the growth could be given.

On May 4 the swelling of iris and cornea was noted to have increased since last examination.

Two weeks later, on May 17, the child was admitted to the Hospital for Sick Children, Great Ormond Street, under the care of Mr. Gunn, to whom I am indebted for the following notes of the case, and for the specimen.

History.—The patient's father died of acute phthisis seven months ago. The mother is in good health. Patient is the fourth child, and was born at full term. She has been weakly and ailing since she was a month old. Has been bottle-fed.

It is not certain for how long the mother had noticed anything the matter with the child's right eye. There has been discharge from both ears for some months, and a "breaking out" on scalp and ears.

On Admission the child was badly nourished; but presented no good evidence of hereditary syphilis. Protruding through the inner part of the right cornea was a fungating mass springing apparently from the iris.

Although the child had had for some time antisyphilitic and tonic treatment, her condition had steadily deteriorated, and the growth in the right eye had increased.

May 31. Right eye enucleated.

The patient improved after the operation, was less restless, and took food well.

She left the Hospital on June 11, and efforts to ascertain the subsequent history of the case have proved unsuccessful.

The eyeball was carefully hardened whole in Muller's fluid, and then divided into two lateral halves for examination.

Naked Eye Examination.—The eyeball is somewhat shrunken, measuring 17 mm., transversely and vertically. The cornea is thickened; at its inner and upper part near

the periphery is a large perforation, probably the site of the incision for iridectomy, through which protrudes a greyish irregular mass, continuous with the iris and ciliary body (*vide* Pl. II, Fig. 2). The iris is enormously but unequally thickened in its whole extent. The thickening is greatest in the lower part, and here the A.C. is almost entirely obliterated. The new growth to which this thickening is due involves the ciliary processes, so that at the lower part they are quite unrecognisable; at the upper part, however, though swollen and altered in shape, they are still distinguishable. The lens has disappeared, but traces of the capsule are visible.

The vitreous is rather too firm, and unusually adherent to the retina, otherwise there are no changes observable in the posterior part of the eyeball.

Microscopical Examination.—The new growth which involves, and to a great extent replaces, the iris, exhibits the usual histological characters of tubercle, very well marked. The giant cells are numerous and large, and are visible in sections of the iris, ciliary body, and the prolapse through the cornea. The small cells which form the larger part of the growth extend posteriorly as far as the hinder limit of the ciliary processes.

There are several areas of necrosis to be seen in the sections.

The pigment layer of the iris and ciliary processes is to a large extent destroyed, though traces of it remain, and are in some places included in and surrounded by the new growth.

The choroid coat, posterior to the ciliary processes, shows scattered islands of small cell accumulation, but there is no general thickening or infiltration. The optic nerve close to the eyeball shows a slight increase of staining nuclei; the papilla is a little swollen, and close to one of the large branches of the central artery is some small cell accumulation in the nerve fibre layer. The retina generally appears healthy, but anteriorly, close to the ora serrata, has undergone some alteration, the innermost layers, down to and including the inner granules, being irregularly thickened and infiltrated with lymphoid cells. From this portion of the retina small new vessels pass inwards into the anterior part of the vitreous, which here shows a moderate amount of cell infiltration; this, however, is confined to the anterior part. The lens capsule is adherent to the iris; the lens itself has entirely disappeared.

The cornea close to the site of perforation is much thickened; the new growth continuous with that of iris and ciliary body extends completely through it, but the cells are also invading the cornea at the margins of the perforation, passing in between the lamellæ for a short distance, and to a greater extent between the posterior layer of the cornea and Descemet's membrane.

Careful staining and examination of sections of the iris and prolapsed mass, both by Mr. E. T. Collins and myself, failed to discover the tubercle bacillus. Several different methods of staining were employed.

Remarks.—There seems little room for doubt that the above are instances of primary tuberculosis of the eye; still it must be remembered that neither case has passed those tests which by some writers are considered necessary to entitle a growth to be called tubercle, I mean (1) inoculation of animals and production of tuberculosis in them, and (2) the discovery in the affected tissues of Koch's bacillus. The first test could not be employed, for obvious reasons.

It is possible that in Case 2 tubercle pre-existed elsewhere, and unfortunately we are without the after-history.

Both patients were young children, with a strongly tubercular inheritance; improvement began almost immediately after the removal of the eye, though only in Case 1 are we able to say that the improvement continued.

Case 1 seems to indicate that by the timely removal of an eyeball, affected by tubercular disease, we may hope to avert general tuberculosis.

Deutschmann* has published a very similar case in which six years after the operation the child was in perfect health.

Case 2 exemplifies the rule laid down by some writers, that if in cases of tubercle of the iris, operative treatment be decided upon, nothing short of enucleation should be

* Arch. f. Ophthal., xxviii, 1, p. 317.

performed. In many cases, however, in an early stage, iridectomy and the removal of nodules upon the iris may be advisable and useful for diagnostic purposes.

A not inconsiderable number of cases of primary tuberculosis of the eye have been published during the last few years. Nearly all have been in children, and a noticeable feature in the cases has been the frequency with which a history of tubercular disease in one or other parent has been obtained. Some cases occurring in adults were published by Haab in Graefe's Archives, 1879, and more recently one by Neese in a man æt. 33.

SYPHILITIC DISEASE OF THE EYELIDS.

By J. Hutchinson, Junr.,
Ophthalmic Surgeon to the Great Northern Hospital, and Clinical Assistant at Moorfields.

Primary Chancres.—The occasional occurrence of indurated sores of the palpebral conjunctiva is thoroughly well recognised, their proportional rarity being indicated by Boeck and Sturgis's figures (7 to 4000 cases of syphilis). There is as a rule but little difficulty about the diagnosis, and the few cases I have seen presented no unusual features. It is, however, interesting to note, that in the cases of infection through a sore of the eyelids or conjunctiva, the chancre usually presents a fairly typical induration, and a bubo which conforms to rule in being indolent, non-inflammatory, and practically painless. As is well known, infecting chancres on other parts of the face, *e.g.*, the chin, lips, or cheeks, are liable to present abnormal signs, that is to say, the induration may be very ill defined, the amount of pus-crust considerable, and the bubo quite atypical. In two or three cases of these chancres I have seen a large, painful, and tender bubo in the sub-maxillary region, differing markedly from the common indolent glandular enlargement met with in the groin. The glands which are first affected in the case of chancre of the eyelids are the pre-auricular and sub-maxillary, thus proving that the lymphatics from this region run towards the parotid in at any rate a number of cases. Here pathology comes to the aid of anatomy, since neither in Mascagni's plate of the lymphatics of the face nor in Quain's description is this course indicated. Sappey, however, has pointed out that whilst the deep lymphatics of the eyelids accompany the facial vein and end in the sub-maxillary glands, the superficial ones pass with the temporal vein to the parotid or pre-auricular lymphatic glands.

Mackenzie, Hirschler, Desmarres, and others, have recorded examples of soft or non-infecting chancres of the eyelids, and this is interesting, since hardly anywhere else on the face has their occurrence been noted. Galezowski's case is a clear one of contagion conveyed by the finger of the patient, who had at the time a soft sore on the penis. Probably this is the usual origin of both hard and soft sores in this region, and one can only explain the preference shown by the latter for the conjunctiva, by the delicacy of the latter compared with the skin of the rest of the face. In the case quoted by Hirschler, infection was conveyed to the eye of a surgeon whilst syringing a chancre on his patient's penis. The sore was situated at one commissure, and healed under purely local treatment.

Case of Ulceration of the Margin of the Eyelids during the Secondary Stage.

A woman, æt. 55, came under my care at the London Hospital for a severe general syphilide. The history was clear as to her having contracted the disease through a chancre of the tongue (the source of infection could not be discovered), and in the third month after the development of the primary sore both upper eyelids became ulcerated. The sores were multiple, superficial, and situated just above the row of eyelashes, so that it was not a true ciliary blepharitis. Under treatment they healed fairly quickly, leaving distinct scars, and for some time the eyelids remained somewhat swollen. There was also some dusky pigmentation of the lids, the eruption on the body being a strongly pigmented one. Previous to the syphilis she had never had any affection of the eyes or lids, and at the time the latter became ulcerated typical sores developed at the angles of the mouth.

Hirschler and others have recorded cases in which the secondary ulceration involved the hair-bulbs, and resulted in linear scars along the margin of the lid with complete loss of eyelashes in the affected region. This, of course, is quite distinct from the shedding of eyelashes which often

accompanies syphilitic alopæcia, and which is nearly always followed by their perfect restoration.

Case of Relapsing Chemosis occurring during the Secondary Stage.

The following case seems worthy of being noted, although its connection with the syphilis from which the patient was undoubtedly suffering at the time may be disputed.

William M., a fairly healthy looking man æt. 26, contracted syphilis, and was treated for a short time for various secondary phenomena with mercury. About seven months later he became liable for the first time to acute attacks of chemosis in the right eye, without other lesion. The conjunctiva would rapidly swell, the chemosis attaining its maximum in the course of 24 hours or less, and then gradual subsidence would follow in the next few days. There was nothing in his occupation to cause irritation of the eye, nor was there anything erysipelatous in its nature, for the rest of the face was never affected. He had never had erysipelas. He attended at Moorfields during the third attack, and scarification was tried on one occasion, though looking at the fact that complete subsidence had previously occurred in the other attacks, it seemed hardly necessary. It should be mentioned that he was still suffering from secondary ulceration of the tongue. The course of the affection, rapid onset, complete subsidence, and tendency to relapse after a few months' interval, somewhat resembled that of herpes of the penis, which is well known occurs not infrequently in syphilitic subjects, although it is not confined to them.

Relapsing Ulceration and Infiltration of the Upper Lid due to Tertiary Syphilis.

Elizabeth A., æt. 36, came under my care at the Great Northern Hospital on August the 9th, 1887, with general thickening of the upper eyelid on the right side. There was some conjunctivitis, but the universal thickening of the lid was the chief feature, and although there was then no isolated gumma and no ulcer the suspicion at once arose of tertiary syphilis. I found that she had suffered from secondary syphilis some three or four years previously, and had for many months been under

specific treatment for one or other tertiary symptoms. She was in fact, one of those patients who may be described as being "saturated with syphilis," and in whom treatment with mercury and iodide only serves to relieve the symptoms and not to prevent the occurrence of fresh ones. She was a florid and rather stout woman, who had previously been rather intemperate.

She was put under 5-grain doses of iodide of potassium and an ointment containing mercury and extract of belladonna. These measures were only partially successful, and by the beginning of November a small ulcer had developed along the ciliary margin of the lid. The liq. hydrargyri perchloridi was added to the iodide in the usual 1-drachm dose, and iodoform applied daily to the ulcer. By November 24th, a typical gumma in the tarsal cartilage ulcerated through on the conjunctival side. I was very anxious to promote rapid healing, fearing the occurrence of severe entropion, but was at a loss what addition to make to the treatment (I have not mentioned all the different remedies which had been tried). Fortunately the process now took a turn for the better, the daily application of iodoform (1 part to 4 of starch) to the bottom of the ulcers appearing to have the best effect. By January 13th, the ulcers were quite healed on the lid. A gummatous node had been for some time present on the top of the head, and an ulcer of the palate which threatened to perforate, but which at last healed under treatment with iodoform and a weak solution of chromic acid. At the beginning of February the upper lid again became greatly thickened, and a fresh gumma formed which ultimately broke and healed with destruction of several of the eyelash-follicles. All this time the patient was steadily taking iodide of potassium or mercury. By May 1888 (nearly eight months after the first affection of the eyelid), the healing appeared to be permanent, but one would not be surprised if a further relapse occurred. An obstinate form of syphilitic lupus of the upper lip has lately developed, but has yielded to some extent to mercurial inunction. It should be mentioned that there had never been any ocular symptom of syphilis, and that the conjunctiva, although not infrequently inflamed, had shown nothing of a specific nature, the disease being practically confined to the upper lid.

The extreme obstinacy of tertiary disease of the eyelids, which is illustrated by this case, has been commented on by most writers on the subject. De Wecker ("Traité d'Ophtalmologie," vol. i, p. 72) relates the case of a lady who came under treatment for a slightly inflamed swelling in the upper lid, which he at first supposed to be a chalazion, but whose nature was made clear by the fact that she had previously suffered from tertiary syphilitic sores of one thigh, and at the same time from a similar swelling of the same eyelid.

An erroneous diagnosis had then been formed, and the gumma freely incised, and it was only after energetic internal treatment with mercury and chloride of gold had been employed that healing took place. De Wecker advised the same treatment, but it was irregularly carried out, and the swelling continued to increase. The use of bichloride of mercury and Zittmann's decoction for two months was successful in reducing its size, but a third relapse occurred, and a large gummatous ulcer resulted. Finally this healed, and a small fistula through the lid which was left by it was cured by the application of nitrate of silver. Besides the tendency to relapse this case agreed with the one I have narrated in other particulars. The final result was much more favourable as regards the scar, &c., than the size of the gummatous ulcer had made one fear it would be, and the disease was accompanied by much more pain and irritation than are usually present in cases of simple tertiary syphilitic sores.

The mistake between a syphilitic gumma of the eyelid and a chalazion appears to have been made so frequently, that one must admit the diagnosis to be sometimes difficult. Usually, however, the chalazion is much more rounded and defined than the gumma, and the œdema and infiltration of the lid are much more pronounced in the latter. Sometimes in the late secondary stage a superficial infiltration of the conjunctiva occurs, which may go on to ulceration if not treated, but which rapidly clears off under mercury.

This "syphilitic tubercle," of which Mr. Vose Solomon records two cases, is usually easy to diagnose, on account of its brownish-red pigmentation, and the occurrence of other tubercles on the face at the same time.

Prof. Taylor, in the "American Journal of Medical Science" for 1875, p. 370, narrates two interesting cases of syphilitic infiltration of the caruncles. Both were in adult men, in one case two years, in the other three years after the primary attack of syphilis. The caruncles became symmetrically enlarged, but this gummatous infiltration (for such there is no doubt it was) was unaccompanied by pain, such as is almost always met with in the similar affection of the eyelids. In one case specific treatment led to absorption, but unfortunately this went on to complete atrophy of the caruncles with resulting epiphora. The result of the other case was somewhat singular; the patient's friends persuaded him that only a "specialist" could treat an affection of the eyes properly, and he went to an ophthalmologist, who diagnosed cancerous growth, and freely excised both caruncles and the surrounding conjunctiva. A plastic operation only increased the deformity, and complete epiphora persisted. He was at the time suffering from rupia of the forehead, so that the mistaken diagnosis was hardly to be excused.

An interesting case is recorded by Mr. Charles Lee, in the "Lancet" of October 31, 1885. The patient was a labourer, aged 44, who had gone through the secondary stage of syphilis some twenty years previously, but who had been since free from symptoms until Christmas, 1884, when his throat became ulcerated, and this progressed until a large part of the soft palate and uvula were destroyed.

In July, 1885, his right eye became inflamed, and when he came under care the lower lid was thickened; on everting this a somewhat extensive ulcer was exposed, involving the caruncles and adjacent conjunctiva. Its edge was thickened but not very hard, and the surface

was covered by scanty thick yellow pus. Pre-auricular glands not enlarged.

It was thought at first that the lesion was a "chancroid of the conjunctiva," and hence only local treatment (nitric acid, followed by iodoform and glycerine) was prescribed. Subsequently, however, the tertiary nature of the ulcer was recognised, and mercury (one-sixteenth of a grain of the bichloride with 5 grains increasing to 20 of iodide of potassium) ordered to be taken three times a day. Under this treatment the ulcers both of conjunctiva and palate rapidly improved, and were quite healed in six weeks' time. In reading the records of similar cases one is struck with the large proportion in which the palate was ulcerated about the same time as the eyelids. Acquired syphilis is much more often the cause than the inherited disease, although a few examples from the latter are to be found. Hirschler narrates one case in a girl aged 13, in whom both lids were swollen and infiltrated, the affection yielding to specific treatment. The diagnosis of inherited syphilis was made almost certain, from the fact that a few weeks previously both her knees had become spontaneously affected with chronic indolent synovitis.

The following conclusions may be derived from the study of recorded cases and those which I have seen:—

1. Late secondary and tertiary affections of the eyelids are met with most commonly in those who have suffered severely from their syphilis, especially those who have had rupial or tubercular skin eruptions or ulcers of the palate. This fact of course helps materially in the diagnosis.

2. The upper lid is more liable than the lower one to be affected, though both are not infrequently involved.

3. The tertiary ulceration (gummatous) of the lids is more often met with in women than in men.

4. Not only do many cases of the latter resist specific treatment in a marked degree, but they show a strong tendency to relapse after being apparently cured.

5. As a rule syphilitic ulcers and gummata of the eye-

lids are more painful than those met with on other parts of the skin.

I suppose this feature must be ascribed partly to the very free innervation of the eyelids, and partly to their constant motion, although gummatous ulceration of the tongue (which is at least as mobile) is not as a rule so painful.

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Windsor. British Medical Journal, June 3, 1865. (Seven cases, chiefly of late secondary ulceration.)

Hock. Wiener Klinik, 1876, p. 65.

Streatfeild. British Medical Journal, September 30, 1882. (One case of chancre of the inner canthus and one of tertiary disease of the lacrymal gland.)

Lawson. Lancet, 1865, I, p. 478.

ATROPINE IRRITATION.

By E. Treacher Collins,

Curator of the Museum.

I will first give the details of 18 cases of irritation caused by the use of atropine to the eye, collected while I was house surgeon at the Moorfields Hospital, and for the use of which I have to thank the members of the staff. I will then proceed to summarize and discuss them.

Case I.—John W., æt. 59.

August 26, 1881. Extraction of cataract from right eye; slight atropine irritation from drops used some days after the operation.

July 17, 1885. Extraction of cataract from left eye; duboisine used as a mydriatic during after-treatment; no irritation.

Jan. 8, 1886. Opaque membrane removed from left eye with a Tyrrell's hook. Gut. Atrop., gr. iv to the ounce, ordered three times a day.

Jan. 25. Lids of left eye swollen, stiff and red; atropine irritation. Father and grandfather both gouty; has never had an attack himself.

Case II.—Stephen C., æt. 60.

Came first as an out-patient on March 16, 1886, with an abscess of his left cornea; the edges of his lids were everted.

March 20. L. hypopyon formed; lower canaliculi slit on both sides. Ung. Atrop., gr. iv to the ounce, ordered twice a day to the left eye.

March 29. Redness and dryness of the skin of both lids on the left side this morning, extending downwards to the cheek, and inwards towards the bridge of the nose.

Ung. Atrop. omitted. Says his father suffered from gout; has never had it himself.

March 30. Skin brushed with solution of nitrate of silver, 3 gr. to the ounce.

April 3. Injection of skin much less.

Case III.—Sarah M., æt. 79.

Jan. 20, 1886. Extraction of cataract from right eye; subsequently Gut. Atrop., gr. iv to the ounce, used once a day for a week. No irritation produced.

March 27. Some spasmodic entropion of right lower lid; membrane in right eye cut with a Knapp's knife; Gut. Atrop., gr. iv to the ounce, one drop at the time of operation, and one 2 hours later. Six hours after operation, redness, swelling, and stiffness of lids, and injection of conjunctiva, evidently atropine irritation.

April 3. Atropine has not been used since, and irritation has subsided. Entropion still continues. Patient has had slight attacks of rheumatism.

Case IV.—Mary S., æt. 59.

Dec. 30, 1885. Cataract extracted from left eye. Gut. Atrop., gr. iv, c̄ Cocaina gr. x to the ounce, used after operation; no irritation produced.

March 30, 1886. Lids of both eyes became inflamed about three or four days ago. Has been using Ung. Atrop., gr. iv to the ounce, and Fotus Belladonnæ; lids are now swollen and red; conjunctiva is injected, and there is slight muco-purulent discharge. Atropine and belladonna stopped.

April 17. Swelling of lids and injection much less. Has never had any rheumatism or gout; father has had rheumatic fever.

Case V.—Archibald M., æt. 71.

Jan. 30, 1875. Cataract extracted from left eye; atropine made up with boric acid, ordered the next day, and followed by considerable swelling of the lids; eye ultimately suppurated.

Jan. 24, 1879. Cataract extracted from right eye; atropine ordered the following day to be used night and morning; no swelling of lids noted, but conjunctiva remained irritable and injected for some time; remained in the hospital for a month.

July 22, 1879. Membrane in right eye needled, and atropine applied twice, followed by redness and swelling of eyelids and cheek.

May 25, 1886. Right eye needled a second time; after two drops of Gut. Atrop., gr. iv to the ounce, both eyelids were

swollen and œdematous, and the whole face flushed, swelling extending across nose to lids of the other eye, and to both cheeks.

He has had several attacks of gout in both great toes, the last attack 15 months ago. No history of gout in his family.

May 30. Face and eyelids desquamating.

June 3. Has an acute attack of gout in his right great toe.

Case VI.—George G., æt. 56.

Nov. 20, 1884. Cataract extracted from right eye.

Nov. 21. Gut. Atrop., gr. ii to the ounce, ordered once a day to right eye.

Nov. 26. A little puffiness and scaliness of lids.

Nov. 27. Great toe inflamed with gout; has had an attack before.

Nov. 28. Atropine irritation, lid a little thickened and eczematous; conjunctiva red.

Dec. 1. Ung. Atrop., gr. iv to the ounce, three times a day to right eye.

Dec. 3. Lids puffy and eczematous; considerable injection and chemosis of conjunctiva.

Feb. 10, 1887. Cataract extracted from left eye.

Feb. 19. One drop of Gut. Atrop., gr. ii to the ounce, dropped into eye yesterday; atropine irritation this morning; swelling of lids, and conjunctival injection.

March 5. Duboisine also caused irritation. Swelling has now quite subsided.

Case VII.—Sarah R., æt. 64.

Jan. 11, 1887. Cataract extracted from her right eye.

Jan. 24. Gut. Atrop., gr. ii to the ounce, ordered twice a day.

Jan. 28. Conjunctival injection, swelling and redness of lids, atropine irritation. Gut. Hyoscinæ, gr. ii to the ounce, ordered twice a day.

Jan. 29. Slight spasmodic entropion of lower lid.

Feb. 12. Swelling of lids has subsided, patient using hyoscine all the time; pupil dilated, some posterior synechiæ. Entropion cured by application of a piece of bent sheet-lead. No history of rheumatism or gout, acquired or hereditary.

Case VIII.—Catherine P., æt. 69.

Jan. 7, 1887. Cataract extracted from her right eye.

Jan. 12. After one drop of Gut. Atrop., gr. ii to the ounce, yesterday, there was some swelling of the lids and redness, also conjunctival injection, which has increased to-day after a second drop.

Jan. 14. Gut. Hyoscinæ, gr. ii to the ounce, ordered twice a day.

Jan. 16. The swelling of lids, and other symptoms of irritation caused by atropine, have quite subsided while patient has been using hyoscine.

Jan. 19. Gut. Atrop., gr. ii to the ounce, ordered twice a day to the left eye.

Jan. 28. Has been using the atropine as ordered to the left eye, without any symptoms of irritation until to-day; now a little redness and puffiness of the lids.

The right, to which hyoscine is being used, is quite quiet. No history of gout or rheumatism, acquired or hereditary. Gut. Duboisiæ, gr. ii to the ounce, ordered three times a day to right eye.

Jan. 31. Considerable irritation, with swelling and redness of lids of right eye.

Case IX.—Jane J., æt. 65.

Was admitted Jan. 17, 1887. Patient has had several attacks of gout; father and brothers were all gouty.

On Jan. 18 cataract extracted from right eye, after which patient used Gut. Atrop., gr. iv to the ounce, for more than a week without its producing any irritation. She was readmitted on March 24, 1887, and a membrane in her right eye was needled, and atropine again used without any ill effects.

On May 3, 1887, she was readmitted with some iritis. Gut. Atrop., gr. ii to the ounce, was put into both eyes three times, and the next morning the lids were very much swollen, more on the right side than the left; the swelling and redness extended to the cheeks and neck; the atropine was omitted and the symptoms subsided; on June 5 she had an acute attack of gout in the great toe.

Case X.—Georgina I., æt. 23.

Feb. 24, 1888. Left eye aphakia after the absorption of a lamellar cataract; one drop of Gut. Atrop., gr. ii to the ounce, put in.

Feb. 25. Atropine irritation chiefly conjunctival; membrane in left eye needled.

Feb. 26. Hypodermic injection of the Nitrate of Pilocarpine, one-sixth of a grain to left temple.

Feb. 27. Left eye quiet; patient says that half an hour after the injection she went to sleep, and when she woke up the eye was quite easy, and she was perspiring very much. Ordered ℞. Pilocarpiæ Nitratis, gr. ii; Atropiæ Sulphatis, gr. i; Aq. Destillatæ, 1 oz., to be used as drops three times a day to left eye.

Feb. 28. The above drops have now been used three times without causing irritation; pupil is semi-dilated. Ung. Atrop., gr. iv to the ounce, to be rubbed into left forearm three times a day.

March 1. There is some puffiness of the lids of the right eye this morning; on the left forearm where the ointment has been rubbed in is a red papular rash, which patient says itches slightly. Hypodermic injection of Pilocarpine, as before, to the left forearm.

March 2. After injection yesterday, irritation of arm subsided considerably, but is not so well again this morning. Hypodermic injection repeated.

March 3. Irritation of arm much less.

Case XI.—Maria B., æt. 36.

Feb. 8, 1887. Catarrhal ophthalmia and keratitis in both eyes. Gut. Atrop., gr. ii to the ounce, ordered twice a day to both.

Feb. 17. Lids of both eyes swollen and red; atropine irritation. Patient says she has had several attacks of pain and swelling with redness of skin in her left great toe. Atropine stopped, and Gut. Hyoscinæ, gr. ii to the ounce, ordered twice a day.

Feb. 24. The irritation, which was chiefly of the skin, not of the conjunctiva, has quite subsided since patient has been using Hyoscine.

Case XII.—John H., æt. 81.

Nov. 1885. Right eye. Cataract extracted, after which atropine was used, and caused irritation.

Feb. 23, 1888. Left eye. Cataract extracted.

March 4. Has some swelling of lids and redness of left eye this morning; atropine irritation; has only had two drops of Gut. Atrop., gr. ii to the ounce, one yesterday and one the day before.

March 7. Has had Ung. Atrop., gr. iv to the ounce, rubbed into his right forearm twice a day since March 5. Now a red papular rash of the skin, which patient says itches considerably, and which was faintly present yesterday. Hypodermic injection of one-sixth of a grain of Nitrate of Pilocarpine to right forearm.

March 8. Arm less red; still large papules; patient complains of its itching at night.

March 10. There are some little pustules on arm now. Says he has never had any rheumatism or gout. Hypodermic injection repeated.

March 11. After injection, rash on arm considerably less red and irritable.

Case XIII.—Mary T., æt. 24.

Dec. 1, 1886. Interstitial keratitis of right eye three weeks. Ordered Ung. Atrop., gr. iv to the ounce, four times a day, and Fotus Belladonnæ to be used frequently.

Jan. 22. Œdema of lids of right side.

Feb. 2. Œdema of lids and infiltration have increased; atropine irritation. Ung. Atropiæ and Fotus Belladonnæ omitted.

Feb. 5. Irritation of the lids still considerable; has been keeping the eye bandaged up.

Feb. 23. Irritation has subsided. Gut. Hyoscinæ, gr. ii to the ounce, ordered twice a day.

Feb. 26. The day after using the Hyoscine drops, lids became swollen and inflamed as before. She has never had rheumatism; has had a stiff elbow all her life; her father has rheumatism; the swelling of lids is more œdematous than usual in atropine irritation; the conjunctiva is not affected.

April 6. Gut. Duboisiæ, gr. ii to the ounce, ordered. After one drop face and lids commenced to swell.

Case XIV.—Charles B., æt. 23. Lamellar cataracts in both eyes.

July 23, 1885. Left eye needled, after which Gut. Atrop., gr. iv to the ounce, ordered three times a day.

July 27. Left eye, curette evacuation; some swelling of lids and redness, also conjunctival irritation due to atropine.

Jan. 21, 1886. Left eye, membrane needled; after the application of Gut. Atrop., gr. ii to the ounce, three times irritation commenced.

July 29, 1887. Ung. Atrop., gr. iv to the ounce, to be rubbed into right forearm three times a day.

July 31. A papular rash has appeared this morning on the forearm where the ointment was rubbed in; ointment omitted.

Aug. 1. Rash more marked; patient says "it itches." Patient has never had rheumatism or gout; mother suffers from gout, grandmother from rheumatism.

Aug. 4. Hypodermic injection of atropine administered to posterior surface of forearm.

Aug. 5. A crop of papules has appeared similar to those produced by the ointment around the seat of the injection.

Aug. 10. Rash on arm is fading and becoming scaly.

Case XV.—Harry C., æt. 27.

May 21, 1885. A stout pale flabby man. Left eye; iritis nine days; gonorrhœa 12 months ago, followed by rheumatism in the knees and inflammation in the left eye; Ung. Atrop., gr. viii to the ounce, ordered every two hours at first, and afterwards gr. iv to the ounce three times a day.

June 11. Slight atropine irritation of left eye.

June 13, 1887. A fresh attack of iritis in left eye; Ung. Atrop., gr. iv, c̄ Cocaina gr. x to the ounce, ordered three times a day.

June 16. Lids of left eye swollen, red, and scaly; conjunctival and ciliary injection. Father has had rheumatism (? gout).

June 27. Gut. Hyoscinæ, gr. ii to the ounce, two drops applied yesterday; lids became swollen and inflamed. Gut. Duboisiæ, gr. ii to the ounce, have also been tried, but produced the same result.

Case XVI.—George B., æt. 21.

Jan. 29, 1887. Eyes first became inflamed three years ago, and again lately. Right, corneal nebulæ; left, corneal nebulæ and keratitis. Maternal grandfather "died of rheumatic fever," æt. 91, was crippled with it for 12 years, his feet used to swell and become red. Mother has had rheumatic fever, and father rheumatism; has five brothers and two sisters; one brother has had rheumatic fever; has not had any rheumatism or gout himself. Ordered hot boric acid fomentations and Gut. Atrop., gr. iv to the ounce, three times a day.

Feb. 19. Ung. Hydrarg. Oxid. Flavi c̄ Atropia ordered three times a day.

Feb. 23. Eczematous condition of lids, slight swelling; conjunctiva not affected; atropine irritation.

Case XVII.—John C., æt. 65.

July 27, 1887. Catarrhal conjunctivitis and small clear ulcers of both corneæ. Gut. Atrop., gr. ii to the ounce, ordered three times a day to both eyes.

Aug. 10. Ung. Atrop., gr. ii to the ounce, ordered three times a day to both instead of the drops.

Sept. 12. Lids swollen and red.

Sept. 21. Lids more swollen; palpebral conjunctiva injected; atropine irritation. Ung. Atrop., gr. iv to the ounce, ordered to be rubbed into forearm on right side night and morning.

Sept. 23. A slight red papular rash is seen this morning where the ointment has been rubbed into the forearm.

Sept. 25. The rash has considerably increased, and is accompanied with considerable irritation.

Case XVIII.—Fred T., æt. 16.

Sept. 21, 1887. Left eye, traumatic cataract.

Sept. 22. Cataract needled. Gut. Atrop., gr. ii to the ounce, ordered three times a day to the left eye.

Sept. 26. Slight atropine irritation of left eye.

Sept. 30. Ung. Atrop., gr. iv to the ounce, to be rubbed into right forearm twice a day.

Oct. 3. A red papular rash on skin of forearm where the ointment has been used.

Oct. 17. Curette evacuation of soft lens matter.

Oct. 24. Gut. Hyoscinæ, gr. ii to the ounce, ordered to left eye three times a day.

Oct. 26. Hyoscine causing irritation; vaseline to be rubbed into left forearm twice a day.

Nov. 2. No irritation from the vaseline. Ordered:—

℞ Pilocarpiæ Nitratis gr. ii,
Atropiæ Sulphatis gr. ii,
Aqua Destillatæ ℥j,

to be used as drops three times a day to left eye.

Nov. 3. Drops causing irritation. Hypodermic injection of a third of a grain of the Nitrate of Pilocarpine into arm.

Nov. 4. Irritation of lids less; injection repeated.

Nov. 7. On omitting hypodermic injection for a day, irritation increased. Gut. Atrop., gr. ii to the ounce, ordered three times a day to left eye, and hypodermic injection administered to left temple.

Nov. 8. No irritation; injection repeated.

Nov. 9. No irritation; injection omitted.

Nov. 10. Irritation on lids.

It will be seen by these cases that atropine irritation, though occurring at all ages from 16 to 81, is commonest in old people: thus of the 18 cases, 11 were over 50 and 7 under.* It will also be seen that the symptoms vary considerably, both in the length of time during which the atropine is used before they appear, and also in their intensity; thus in Case V, after only two drops of atropine, the whole of the face was swollen and œdematous; while Case XVII had been using drops for a month, and then the inflammation was confined to the skin of the lids and conjunctiva; in some cases, again, the irritation is almost entirely confined to the skin of the lids, and in others the conjunctiva suffers most, at times becoming almost granular.

The cause of this affection has been attributed to some imperfection in the drug used. At one time the presence of

* I have recently seen a case in a boy aged 5, under Mr. Gunn's care, at Moorfields Hospital.

free acid in the solution was thought to set up the irritation, but as Mr. Lawson says in vol. vi, p. 120 of these Reports:—"What amount of free acid can there be in the one or two drops which are dropped into the eye from a solution of the sulphate of the strength of gr. i ad aquæ ℥j? and yet when thus diluted I have seen it occasion great irritation."

M. Kroemer of Basil, in the "Corr. Blätt. f. Schweiz. Aerzte," xi, 1881, says he believes the conjunctival inflammation which occasionally follows the use of atropine drops is of septic origin, and thinks there is some connection between it and the fungoid growth which takes place when an atropine solution is kept for some time, and goes on to state that he had not observed it since he had used a carbolised solution of the strength of 1 in 1000.

Mr. Simeon Snell, on p. 340 of vol. i of the "Ophthalmic Review," says he has come to the same conclusion, having used salicylic acid in his solution, but he draws a distinction between those cases that get irritation from prolonged use, and those that get it after the application of only one or two drops. In these latter cases he thinks there is some idiosyncrasy.

Without denying that it is possible to produce a conjunctival inflammation by using a solution of atropine in which there is a fungoid growth, still I have produced irritation with perfectly fresh solutions, also with solutions made up with boric acid and carbolic acid, and with ointments made with vaseline. Moreover, it certainly cannot be solely due to some imperfection in the drug, for if this were so the imperfect solution ought to produce irritation in all cases in which it is used; instead of this we find that there are certain individuals who get it with any sort of solution, and others who can use all solutions with impunity.

Is there then some idiosyncrasy on the part of the patient? Mr. Lawson, in the article already quoted,

says:—"I believe that when atropine acts thus as an irritant, or produces peculiar and distressing symptoms, it is due to some idiosyncrasy on the part of the patient which renders him intolerant of the alkaloid or of its salts, and in some cases of any preparation of belladonna."

If this is so, why should it occur when applied to the eye specially? Does it only irritate at the junction of mucous membrane and skin, or has its mydriatic property anything to do with it? To test this, I had rubbed into the forearms of several patients who got atropine irritation of the eye an ointment made of atropine, gr. iv, vaseline, ʒ i, and in all the five cases in which I used it an irritable red papular rash appeared on the skin; in some cases these papules developed small vesicles on their surface, and in one case they went on to pustules. Case X, in whom I produced this rash on the forearm, had got irritation of the eye from one drop of a two grains to the ounce solution; Case XVII also got a rash on the forearm, and he did not get irritation of the eye until after a month's use of atropine.

In order to prove that this rash was actually caused by the atropine, in Case XVIII I rubbed vaseline into his other arm without producing any effect, and in Case XIV I injected some solution of the sulphate of atropine hypodermically, and around the seat of the injection a crop of papules appeared.

These facts show that atropine irritation is not a purely local affection, and also that cases in which the irritation occurs after a few drops, and those in which it occurs only after a prolonged use, are of a similar nature. We know that atropine or belladonna applied locally has the power of checking secretion; to try whether its action in this way had anything to do with the irritation produced, I, in Cases X, XIV, and XVIII, injected hypodermically a solution of the nitrate of pilocarpine which has the opposite action, that is, increases the secretion of the skin. In all these cases it seemed to have a temporary effect in

relieving the irritation both in the lids and eye, and on the arm the redness became less, and the patients said there was less irritation, but this relief was only transient. In Case XVIII by repeating the injections daily I was enabled to continue the use of atropine to the eye, and so keep the pupil dilated without any irritation. I have also tried drops made of a mixture of pilocarpine and atropine, but without any satisfactory result.

Why should the checking of secretion give rise to irritation? M. von Zehender, in the Bowman Lecture for 1886, says:—"The germs of all living microzoa floating in the air can every moment drop into the open eye, and remain on the surface of the conjunctiva. Hence it cannot be astonishing that one finds in every normal eye plenty of germs of all possible kinds of micro-organisms; of two persons with quite normal conjunctivæ, it has been found one eye almost always contains pathogenic germs (Gifford, "Archiv für Augenheilk.," Bd. xvi, p. 199);" and he goes on to ask: "Why does not then the eye become inflamed every moment?" May it not be that the normal secretion of the conjunctiva (or of the skin, in the case of the lids and arm), is one of the barriers, and when this secretion is checked, as I am supposing it is in some people by the atropine, inflammation results? This theory would lead us to suppose that there are certain individuals in whom the secretion of the skin or conjunctiva is checked very rapidly by atropine; others in whom it is only stopped after a prolonged use of the alkaloid, while in the vast majority it is never entirely checked. Again, we might suppose that sometimes the necessary microbes were absent or not present in sufficient quantities to cause inflammation. By these means we could explain the extreme variability in the onset, and intensity of the symptoms at different times and in different cases.

In Case II there was slight ectropion of the lower lids, and the puncta were everted, so that there was some obstruction to the flow of tears down the lacrymal ducts.

Case III, when first in the hospital, used atropine for a week and had no irritation, the second time she came in she had spasmodic entropion, and so the drainage of the lacrymal secretion was hindered; she on this occasion after only two drops of atropine got irritation. Case VII also had spasmodic entropion.

In these three cases it would seem that the interference with the passage of the lacrymal secretion down the lacrymal canal predisposed them to atropine irritation. By the constant secretion and drainage of the tears the microbes which collect in the conjunctival sac are being washed away; if the drainage be prevented in any way, they will be increased in numbers, and so more ready to set up inflammation.

If the inflammation known as atropine irritation is due to irritation set up by microbes normally present in the conjunctiva, and which are enabled to act in this way by the checking of the normal secretion, ought not the same thing to occur sometimes when the normal secretion is arrested in other ways?

The following case is an example of this:—

Jane W., aged 60, had a cataract extracted from her right eye on July 3, 1886; iritis set in on July 9, and Gut. Atrop., gr. ii to the ounce, were ordered twice a day; on July 14 there were considerable redness and swelling of lids with conjunctival injection. I took the case to be one of atropine irritation. Substituted duboisine for atropine and had the eye untied; the inflammation then subsided. She was readmitted Nov. 23, 1886, and iridectomy was performed on her right eye; the next day the lids were a little puffy and red, one drop of atropine was put in; the second day after the operation the swelling had increased, the eye was then untied, hyoscine was used instead of atropine, and the symptoms of irritation disappeared.

In April, 1887, she was admitted for extraction of cataract from her left eye; in the fundus of her right eye there were seen to be several small scattered punctate hæmorrhages; her urine contained sugar, no albumen, sp. gr. 1025; acid.

The day after the operation on the left eye the lids of both eyes were swollen, and the conjunctivæ were injected though no atropine had been used; this swelling and redness had increased by the next day. When both eyes were untied and the symptoms subsided, atropine was afterwards used, and as long as the eye remained untied no irritation occurred; on two occasions I injected some atropine hypodermically into her arm, but no rash was produced.

Still supposing all this to be as I have suggested, it leaves unexplained why some people are very readily affected and others never; or why in some the secretion of skin or conjunctiva is so quickly checked by atropine. What is the idiosyncrasy? It is not the same as that which renders some people abnormally liable to the toxic effects of atropine, for I have seen dryness of the mouth and even delirium supervene after the application of atropine drops to the eye, but in such cases no inflammation of the lids has occurred, and duboisine and hyoscine, which more often produce toxic symptoms, less often produce irritation of the eyes; thus hyoscine was used in 6 out of the 18 cases, and in 3 only did it produce irritation; duboisine was used in 5 cases out of the 18, and produced irritation in 4 of these.

Nor is it that it occurs only in eyes in which the vitality has been lowered by past or present inflammation, for I have produced it in eyes that were practically normal; the worst attack of atropine irritation I have ever seen was in Case V, and he was a man who had had several attacks of gout; while in the hospital about a week after the irritation in his eye he had a very severe one. This led me to enquire into the arthritic history of these cases, with the following result. The figures are not sufficiently numerous to draw any conclusions from: I will therefore simply give them without comment.

Of the 18 cases:—

4 had had acute attacks of gout.

3 had a family history of gout.

1 had had gonorrhœal rheumatism and a family history of rheumatism (? gout).
1 had had rheumatism.
3 had a family history of rheumatism; and
6 had no history of gout or rheumatism either acquired or hereditary.

ON THE TREATMENT OF SUPPURATION AFTER EXTRACTION OF CATARACT.

By E. Treacher Collins.

When suppuration of the cornea has once commenced after extraction of cataract, so rapid is its progress that in spite of treatment panophthalmitis generally follows; and even in those cases where the suppurative process has been checked, so much damage has generally resulted, that in most the eyes become disorganised and shrunken.

Von Gräfe, in 1863, in some clinical remarks on a case of extraction of cataract (which have been translated by H. Derby), after deprecating the use of ice compresses, of leeches, and of venesection, recommends the application of what he terms "a constrictive bandage," in which the orbit is packed plentifully, and a flannel roller passed three times over the eye in question,—and also of warm aromatic lotions. The former of these measures he says "I cannot praise too highly." He seems to have attributed the suppuration to want of adaptation of the two edges of the wound, and it was this which he hoped to effect by the use of firm pressure.

Having tried this form of treatment in several cases, and having in all of them found that the suppurative process instead of being checked rapidly ran on to panophthalmitis, and believing the suppuration in the majority of cases to be due to infection, and not to want of adaptation of the edges of the wound, I was induced to try the very opposite plan, viz., as soon as any sign of suppuration showed itself to leave the eye untied, and have it frequently bathed with a tepid antiseptic lotion, only a piece of lint kept moist with the lotion applied as a blinker over the eye. By these means, instead of keeping the discharge pent up in contact with the wound, a free escape is permitted, and the wound kept clean by

frequent bathing with the antiseptic lotion; at the same time I endeavour to combat any marasmic tendency by extra diet, stimulants, tonics, and by allowing the patient to get up out of bed and sit about.

The first case I treated in this way was a man in whom suppuration set in after the removal of a diabetic cataract (Case No. XXXIV in the table of suppuration cases after extraction, published in Part I of vol. xii of these Reports). This case recovered, but with a closed pupil; the tension, however, remained normal. Of six other cases I have treated in this way, in three the suppurative process has been checked, though in two of these the globes ultimately shrank; in the other three the suppurative process continued unabated.

Mr. Nettleship, on June 23, 1886 (in Case XLI), applied the galvano-cautery to a yellow line of infiltration which was commencing along the margin of the cornea, freely and deeply across the whole length, but not opening up the anterior chamber; the eye was then treated as above stated. There was a striated haze of the cornea after this, which subsequently cleared up, and on August 18 the eye was quiet, and the tension normal, but the pupil blocked with a dense membrane. On October 30, 1886, an iridectomy downwards was performed with a Tyrrell's hook, and on November 6, V. $\bar{c} + 10 = \frac{16}{24}$, $\bar{c} + 14$ J. 6.

Since that time I have applied or seen the galvano-cautery applied in eight other cases, in all of which the after treatment consisted in leaving the eye untied and using antiseptic lotions. In three of the nine cases thus treated the eye has been saved and good vision ultimately obtained; in three the suppurative process was checked, but flattening of the wound and shrinking of the globe supervened, and in the remaining three panophthalmitis set in.

I will now give in detail the particulars of the other two cases in which good vision was obtained.

Samuel W., æt. 54, was admitted under Mr. Nettleship on May 10, 1887; he had nearly complete cataracts in both eyes, somewhat more advanced in the left than in the right. V. in R. = fingers at 2 feet, projection good; in the L. V. = hand reflex, projection good; pupils normal, and tension normal in both; no lacrymal complication.

May 11. L. extraction with iridectomy, capsule opened with Förster's forceps, operation uncomplicated.

May 17. Slight injection, anterior chamber reformed, haze of cornea at upper part extending from wound.

May 20. Cornea bulged forwards more in one direction than in the other, looking very astigmatic.

May 21. Some grooving of the wound and some iritis, two posterior synechiæ; at the seat of wound this morning noticed for the first time a yellowish-grey nodule, a commencing patch of suppuration. L. Galvano-cautery applied to margin of wound.

May 25. Considerable grooving of the wound and some striped keratitis; suppuration has not extended.

May 28. Injection much less, the patch at seat of wound is more of a grey colour.

June 2. Wound healing; very little injection. V. of L. $\bar{c} + 10 = \frac{6}{60}$, $\bar{c} + 14$ J. 16.

Aug. 10. An opaque capsule, cut with a Knapp's knife, without much result.

Aug. 13. Capsule torn with a needle.

Aug. 18. V. of L. $\bar{c} + 12 = \frac{6}{18}$, $\bar{c} + 16$ J. 4.

Elizabeth E., æt. 64, admitted March 22, 1888, with incomplete cataracts, with a central, white, chalky-looking patch in both eyes. V. in both = J. 19, projection good, pupils normal, tension normal, no lacrymal complication.

March 23. R. extraction with iridectomy, capsule opened with Förster's forceps, operation uncomplicated.

March 24. No swelling, pain, or redness.

March 25. Pain in the side of face and in the eye last night; slight puffiness of the lids this morning, slight purulent discharge, conjunctival injection, purulent infiltration of wound not extending backwards. Galvano-cautery applied to infiltrated margin of wound, eye left untied with blinker soaked in

Lotio Hydrarg. Perchlor. over it, eye bathed with the same lotion every two hours. Unguentum Iodoformi c̄ Atropia made up with Lanoline applied three times a day. Beef tea and wine extra.

March 27. No fresh infiltration of wound; striated keratitis; very slight discharge, good anterior chamber. Two leeches applied to right temple.

March 29. Pupil partly dilated, two posterior synechiæ; swelling of lids and conjunctiva less; wound now looks healthy, still some striated keratitis.

March 31. No pain; injection and swelling less; wound healed. There has been no fresh purulent infiltration, the keratitis is less, aqueous is not turbid, and the pupil is black.

April 7. No injection at all, wound well healed, no keratitis.

April 17. V. of R. $\bar{c} + 12 = \frac{6}{24} + 16$ words of J. 4.

It will be seen that in these three cases the cautery was applied in a very early stage, that is, within a few hours of the first indication of any suppuration starting. In those cases in which the result was not so satisfactory a longer time had elapsed between the onset of symptoms and the cauterisation; and in those cases in which the suppurative process was checked, and in which the globes ultimately shrank, a large portion of the cornea had become infiltrated, and consequently more tissue had to be destroyed by the cautery, the result of which was flattening at the seat of the wound and minus tension. Case L in the table is a typical one of this sort:—

Hannah B., had a cataract extracted from her right eye on November 1, operation uncomplicated.

Nov. 3. Complains of pain; eye not examined.

Nov. 4. Swelling and redness of the lids; considerable yellow infiltration of the cornea. Galvano-cautery applied to the infiltrated portion, eye left untied, and bathed every two hours with Lotio Hydrarg. Perchlor.

Nov. 5. Infiltration has not increased. Ung. Atrop. c̄ Iodoform made up with Lanoline ordered three times a day.

Nov. 8. Part of the cornea that was infiltrated is now grey. V. = p. l.

Nov. 26. Anterior chamber has reformed; cornea clear except at the upper part; coloboma filled with a dense inflammatory membrane.

Nov. 28. T. —3, flattening at seat of wound, V. = p. l.

If a still longer time has been allowed to elapse between the onset of symptoms and the application of the cautery, it is found that not only has a large extent of the cornea become involved, but that the suppuration has extended backwards into the pupil, and then no amount of cauterisation is of any avail; these cases end in panophthalmitis.

All this shows how attentively the early signs of suppuration should be watched for. The first symptom which is likely to attract attention in nearly all cases is pain; this, together with the infiltration of the cornea, often commences some hours before there is any swelling of the lids or discharge; there are, however, exceptional cases in which there is no pain at all. Of course pain may be due to numerous other causes, and patients' ideas with regard to pain vary considerably. Still, I believe it best, if a patient complains of pain after the day on which the operation has been performed, to make a thorough examination of the eye, and to see what the real condition of things is, and this even if the pain has passed off, for in suppuration cases patients frequently give a history of acute pain lasting some hours and then subsiding. If, then, there is seen to be no infiltration of the cornea, the appropriate treatment, whatever it may be, can be adopted with confidence, but if the margin of the wound is infiltrated, the cautery should be applied there and then without delay. I believe if this plan were adopted many eyes now lost by suppuration would be saved.

Assistance might also be gained in detecting suppuration in its early stages by the systematic use of the thermometer in cataract cases. I have not had any experience of it myself, but Mr. Simeon Snell speaks of the value of its use in vol. iii, p. 105, of the "Ophthalmic Review."

ON THE PATHOLOGICAL ANATOMY OF LAMELLAR OR ZONULAR CATARACT.

By J. B. Lawford,

Late Curator of the Museum.

Opportunities of examining crystalline lenses affected with lamellar or zonular opacity are so few, that it seems worth while to record the results of the examination of three lenses (from two patients) which have come into my hands as Curator of the Hospital Museum.

Case 1.—Thomas B., æt. 44, shoemaker, admitted to the Royal London Ophthalmic Hospital under Mr. Nettleship's care on November 5th, 1886.

His history so far as ascertained was that he had never had good sight, that he had been unable to obtain spectacles which were of any assistance to him, and that he liked to shade his eyes, as he could then see better. He thinks his sight has become rather worse in the last 10 years.

He is the fourth child of six; his brother and four sisters have no defect of sight. He is married and has eight children, all of whom are healthy and have good sight.

On admission, V. R. $\frac{6}{60}$, 16 J. L. $\frac{6}{60}$, 16 J. Cornea, pupil, and anterior chamber normal in each. T. n. In the lens of each eye, a fairly dense typical lamellar opacity. Teeth much discoloured and irregular from loss of enamel. History, concerning fits during childhood, not known.

On November 6th the lens of the *right* eye was extracted through an upward section. The capsule was opened by incision near its upper edge, and the lens came out nearly complete. The wound healed well, and patient left the hospital November 24th.

After needling of some opaque capsule in the pupil, the vision of this eye was $\frac{6}{18}$ and 1 J. Patient returned in February, 1888, for operation on the left eye, the sight of which had not altered in the intervening 14 months.

On February 4th, 1888, the lens of the left eye was extracted

in its capsule. This eye also recovered well from the operation, and its vision with correcting glasses was two months later $\frac{6}{24}$ and J. 6.

Both lenses were treated in the same way, *i.e.*, carefully hardened in Müller's fluid for some weeks; transferred to weak and subsequently strong alcohol; embedded in celloidin. Sections were made in an antero-posterior direction; the staining reagents used were hæmatoxylin (Ehrlich's formula), alum-carmine, and cochineal; a number of sections were mounted unstained, and proved more useful than the coloured ones to exhibit the changes described below.

Right lens (fresh). The outline is irregular, the capsule and some of cortical layers are wanting. The anterior surface is marked by the notch cut by the cystitome, the posterior surface by its somewhat greater convexity. The transverse meridians of the lens measure 8 mm. and 8·25 mm. respectively, so that it has a slightly oval shape. The lamella of opacity is plainly visible, is very thin, and exhibits tiny spokes jutting out from it. As nearly as can be ascertained in the present condition, it measures 5·25 mm. transversely. The portion of lens outside the lamella is fairly clear and not very hard.

Microscopical Examination.—The most noticeable feature in the sections of the lens is the difference in appearance between the nuclear and cortical parts (Plate I). Beyond some separation of the fibres and the tearing of the outermost layers which occurred during the extraction, there is no noteworthy change in the cortical layers. The nuclear portion of the lens, however, exhibits very evident change, though as will be seen from what follows, it is doubtful to what extent these changes are pathological, and to what extent artificial, the result of the fluids employed in the preservation of the specimen.

This central area (Plate I) is very similar in shape to the whole lens, and its outlines are almost parallel with those of the lens; it is, however, rather nearer the posterior than the anterior surface; this may be the result of some of the posterior cortex of the lens remaining adherent to the capsule during the extraction. This area measures (in sections made through the middle of the lens) 2·325 mm. antero-posteriorly, 5 to 5·25 mm. transversely. It contains very numerous, irregularly shaped

spots and patches, which vary considerably in size, and are generally larger and less closely packed in the central part than towards the boundary of this area. These small spots become coloured by staining fluids to about the same degree as the lens fibres; they have an almost homogeneous appearance, and look like particles of very fine sand scattered over the lens fibres; they are not, however, at all raised above the level of the fibres. These particles exhibit a somewhat concentric arrangement as if following the layers of lens fibres. They vary in size from 0·0125 mm. to 0·025 mm., and their shape is quite irregular, some are circular, others have a dentate border. In unstained sections they are bright and highly refracting. It is very difficult to give a good representation of them in a drawing.

Although this central area is very distinct from the cortical part, there is in this specimen no continuous line separating the two parts of the lens. Here and there, however, are fragments of such a line of demarcation which can easily be supposed to be parts of a lamella in the lens, in the continuity of which there are breaks. Outside this line, especially anterior to it, there are several irregular patches of degeneration in the lens fibres, some of which project towards the cortex, and which perhaps are the representations of the spokes seen during life. They are not evident in Plate I, except on the anterior surface of the dotted area. The boundary line between the central and peripheral areas has a peculiar appearance not unlike that of a twisted rope, and its edges are slightly serrated; it varies in thickness a little, its measured diameter being from 0·0125 mm. to twice that thickness. The lens fibres have a decided tendency to split along this line, as though it formed a layer of degeneration, or of some change which reduced the adhesiveness of the adjoining layers.

In a few sections there is but little trace of this boundary to be seen, while in others it can be traced almost all round the central area, though frequently interrupted by short breaks. At the lower extremity in Plate I, there is an indication of a double line. It becomes coloured to almost exactly the same degree as the rest of the lens. The dots or granules in the central part reach generally quite up to this line, but do not extend beyond it.

Left lens (fresh.) Capsule entire and shape of lens about

normal. Its diameters measure 9 mm. transversely and 5 mm. antero-posteriorly. The lamellar opacity which shows very plainly is as nearly as can be measured 4·5 mm. in width. The rest of the lens is nearly if not quite clear, with the exception of a small opacity on the capsule near the equator.

Although the reagents employed, and the time taken in preparing this lens for examination, were as nearly as possible the same as in the case of its fellow, the results were much less satisfactory. After imbedding in celloidin the lens was divided antero-posteriorly into two halves, and sections of each half were made; those nearest the cut surface were not successful, so that I have no section which passes exactly through the centre of the lens and of the opaque lamella.

Microscopical Examination.—In this, as in the right lens, there is a central area of almost the same shape as the lens, which presents appearances very different from the cortical part. It contains very numerous small spots similar in size and appearance to those described above. The cortical layers of the lens exhibit marked changes which are almost entirely if not entirely due to the action of the hardening reagents. There are in places fragments of a boundary line between the nuclear and cortical areas, resembling in appearance that described in the other lens, but there is apparently much less of it. On its outer side, but close to it are several areas in which the lens fibres appear interrupted by a small nearly homogeneous patch, which projects a little towards the capsule of the lens. These patches, which probably represent the spokes seen on the zonular opacity during life, have not the appearance of post-mortem change, but more closely resemble the rope-like line which divides the nuclear from the cortical area.

The lens capsule and the sub-capsular epithelium exhibit no changes.

Altogether, the results of the examination of this lens are not satisfactory. This is, I think, simply due to the fact that the process of hardening produced considerable change in the lens, possibly on account of the impermeability of the capsule.

Case 2.—Ann C., æt. 30, admitted to the Royal London Ophthalmic Hospital, under Mr. Tweedy's care, on April 27th, 1888, suffering from lamellar cataract.

Unfortunately no particulars were entered in the in-patient notes, as to her history or the acuteness of sight at the date of admission.

The lens of the left eye was extracted on the day of admission, through an upward sclero-corneal incision. The lens capsule was divided at the upper periphery and the lens came away nearly entire. The wound healed well, but there was some subsequent iritis and a secondary operation (needling) became necessary. This was performed on June 12th, and was also followed by iritis, as a result of which the pupil became updrawn.

At her second visit, the sight of the right eye (the unoperated one) was noted as $\frac{10}{200}$; beyond the lamellar cataract there were no changes. The extracted lens was prepared for examination precisely as were those of Case 1.

The outline of the lens is irregular, some of the outermost cortical layers are wanting. The shape of the lens is, however, sufficiently well preserved to enable one to distinguish the anterior and posterior surfaces. The lamellar opacity is very distinctly visible, and looks of large size in proportion to that of the lens. The diameters of the lens as removed are 7 mm. transversely and 3 mm. antero-posteriorly; the transverse measurement of the lamellar opacity is 4·5 mm. The outline of the latter in antero-posterior section, though not quite parallel to that of the extracted lens, would probably be so if the entire lens in its capsule had been obtained for examination. There is, as in the normal lens, a greater convexity in the posterior than in the anterior layer of the opacity. This is more noticeable in sections near the periphery, and less so in those at the centre of the lens.

Microscopical Examination.—The description of the left lens of Case 1 will in the main apply to this specimen. There is the same marked difference between the nuclear and cortical parts, and the transition from the one to the other is quite abrupt. Concerning the cortical layers there is little to be said; there is splitting of the layers in several places, and ragged broken fibres at the surface, but no evident change in the fibres themselves. The nuclear part is filled by large numbers of the particles above described, which particles are generally

most numerous and smallest towards the boundary of this area, and comparatively few in number in the centre of the lens. Their average size is 0·0142 mm.

This area, as already mentioned, is of the unequally biconvex shape of the normal lens, as is indicated in Plate B, Fig. 1, in which the anterior surface is uppermost.

Bounding this dotted area is a line which under the microscope is very like a rope, with twisted strands (*a*, Fig. 1, Plate II). This line is in no section continuous all round the nuclear part. There are numerous breaks in it, as is shown in the drawing, but at these breaks there is no irregularity in the contour of the dotted area; although there is a noticeable tendency in the layers of the lens to split along this rope-like line, it is quite clear that in good sections, such as that from which Fig. 1, Plate II, was made, the appearance is due not to separation of the layers and formation of an empty space between, but to some change in the lens fibres which gives rise to this line. A somewhat similar appearance is often seen in sections of lenses at points where the laminæ are just beginning to separate, or where a very tiny chink has formed between adjoining layers. The impression it gives is one of fine oblique striæ in the lens fibres. The width of this boundary line varies from 0·0125 mm. to 0·0375 mm. in sections near the centre where it is cut as nearly as possible at right angles to its surface; in sections near the equator of the dotted area, in which the bounding layer is cut obliquely, the width is of course rather greater than this. In the length of this line there are several small irregularities, like small knots in a rope, but, as in the rope, the continuity is not broken at these points. There is, however, an absence of the small spurs projecting from the dotted area into the cortical layers, such as were seen in some of the sections of Case 1, nor is there any indication of a double line like that in Plate I at the lower extremity of the nuclear area.

It will be seen by the foregoing description that the three lenses examined present very similar appearances. In all there is a nuclear or central area of moderate size, the outlines of which run a nearly parallel course to those of the whole lens. In the portion of lens external to this

—the cortical layers—there are no changes of any import, a few small patches of degeneration being all there visible. The line of demarcation between the cortical and nuclear areas is abrupt; there is no gradual change from one to the other. At the boundary there are indications of a thin layer, as seen in transverse section in the specimens, (*a* in the drawings) which probably extends all round, and encloses the central area. This lamina compared above to a twisted rope, does not in any of the sections form a continuous line, but exhibits numerous breaks. It seems fairly certain that it represents some definite and considerable change in the lens fibres, and probably constitutes the essential part of the "lamellar cataract." The finely dentated edges of this line suggest that it is composed of lens fibres, but its thickness exceeds that of single fibres of a healthy lens, so that the alteration in structure, whatever it be, probably involves more than one layer of fibres.

In the nuclear area in all three specimens, are large numbers of small irregular dots or particles, arranged more or less in layers which seem to run concentrically, as if following the laminæ.

Whether or no these are entirely the result of the fluids employed in preserving the lenses, in other words whether they are coagulation masses, I am unable to say positively. My impression is that they do to a large extent own this origin, but it is certainly a little remarkable that they are so accurately limited to the central area. It may be, however, that there is some diffuse change in the nuclear part of lenses with lamellar cataract, so that it is affected by hardening fluids in a different way to the cortical portion. I have never found (so far as I remember) changes exactly like these, both as regards their appearance and their arrangement, in healthy lenses, of which I have examined a great many, prepared in precisely the same way. In order to help me to arrive at a decision on this point, I prepared and cut sections of several lenses from both young and aged eyes generally healthy, but in

some instances affected by senile cataract. In two of these, one (normal) from the eye of a child æt. 3 years, and one from the eye of a man æt. 65, the former hardened in weak to strong spirit, the latter in Müller's fluid and then spirit, I found numerous small rather bright dots among the lens fibres. These, which were smaller and more regularly oval in shape, were in the first specimen scattered over the whole of the lens; in the second they were limited to the nuclear area. They were no doubt coagulation products such as Becker has described and figured in his work on the lens.

Regarding the pathology of lamellar cataract but little has been published, and in ordinary text-books of Ophthalmology nothing is said about it. Even in Becker's classical work* no description of lamellar cataract is given. Ed. Jaeger† and Von Graefe‡ were the earliest to record the examination of lenses with lamellar cataract, but no microscopic description is given. These observers found in sections of a dried lens a sharp whitish line ($\frac{1}{3}$ to $\frac{1}{2}$ mm. in thickness) separating "a normal nucleus from an unaltered cortex."

In Arch. f. Ophthal., xxxii, 2, 1886, Deutschmann gives a short account of the examination by him of a lamellar cataract from the eye of a person who committed suicide. He found the nucleus and cortex normal in appearance, but divided by a very definite zone of altered lens substance which ran concentrically round the nucleus, and consisted of altered lens fibres with numerous vacuoles and free droplets of myelin. In small chinks which were visible in this zone there was a finely granular detritus. A little more peripherally there was a second similar but incomplete zone. These zones were of varying thickness, their diameter being greatest at the poles (*see* drawing in Deutschmann's article).

* "Zur Anatomie des gesunden und kranken Linse."
† "Staar und Staar-Operationen." 1854, p. 17.
‡ A. f. Ophth., I, 2, 236.

The most recent and full description of the pathological anatomy of lamellar cataract is one by Beselin in Archiv f. Augenheilkunde, Bd. xviii, Heft 1, 1887. He had the opportunity of examining a lens extracted by Professor Schweigger from a man æt. 64, suffering from zonular cataract. The patient had had an artificial pupil made by iridectomy, and had been able to see sufficiently well till a few months before the extraction operation. His sight was then $\frac{1}{12}$ to $\frac{1}{9}$ in the left eye; the right had been lost many years previously after needling of the lens. After extraction L. V. $= \frac{1}{2}$. Beselin's description and illustrations and mine have several points in common. I had cut and examined sections of the right lens of my Case 1 before Beselin's article was published, and had made out practically all that I have now written. These notes were not published then as I knew that probably the second eye of that patient would be operated upon. As a result of this delay I was fortunate enough to obtain a third lens, and also to have the advantage of comparing my specimens with the description given by the writer just referred to.

In Beselin's specimen the area enclosed by the lamella measured in central sections 5 mm. in its transverse and 2·1 mm. in its antero-posterior diameter. The lamella consisted of a narrow zone, or rather of two incomplete zones, that on the anterior overlapping the ends of that on the posterior surface at the equator; so that there appeared to be a double layer at the margins of the lamellar opacity. These zones the author describes as chinks filled with finely granular material, containing some rounded corpuscular elements, probably fat droplets. In the greater part of its course the borders of this chink were formed by normal lens substance; its cross measurement varied from 0·016 mm. to 0·08 mm.

The nuclear area, enclosed by the zone of degeneration,

contained large numbers of small irregularly-shaped dots closely grouped and smaller at the peripheral part of this area, and larger and more scattered in the centre. These Dr. Beselin thinks are coagulation masses, the result of hardening the lens in alcohol, and thinks that if present during life greater defect of sight would have occurred. He has found similar changes in lenses with senile cataract when preserved in alcohol, but not when kept in Müller's fluid. My experience is somewhat different to his, though in the main we are agreed as to the origin of these masses. Our description and figuring of them are almost identical.

It will thus be seen that so far as our scanty knowledge on this subject will carry us, lamellar or zonular cataract (either name seems to be strictly applicable) consists essentially in a narrow zone of degenerative change in the lens fibres situated between the nuclear and cortical areas. Of the exact nature of this change, we can as yet say but little. It is possible also that the nucleus of the lens within this zone is in some way abnormal in structure. It is to be hoped and expected that future investigations will throw more light upon both these questions. That we have not already a better knowledge is due partly to the rarity with which lamellar cataracts are obtainable for microscopic examination, and partly to the fact that the crystalline lens is one of the most difficult of all tissues to manipulate for such examination. As to the cause of the occurrence of this form of cataract, whether a developmental defect, or some nutritive change in early infantile life, these microscopical investigations do not seem to assist in the determination.

DESCRIPTION OF PLATES.

Plate I.

Drawing of a nearly central antero-posterior section of the left lens of Case 1. The outer fibres are torn and ragged, where they have separated from fibres left in the capsule. The notch in the anterior surface was cut by the cystitome. The central dotted area is shown in its entirety, and also the greater number and closer aggregation of dots near its periphery. At the points *a*, *a*, *a*, are fragments of the rope-like line separating the central and cortical parts; in places (as at the lower extremity), this appears double. On the anterior boundary line are several irregular patches of degeneration in the lens fibres. × 25.

Plate II.

Fig. 1. Drawing of part of a section of the lens of Case 2. The anterior surface is uppermost. The dotted area with its rope-like boundary is well shown, this line being here present in greater lengths than in most of the sections. It has, however, come out rather too distinctly in the print: it should be softer and less evident. × 85.

Fig. 2. Drawing of the cornea, iris, and ciliary body of the eye of Case 2, in Tuberculosis of the Eye cases (p. 152), showing the great thickening of the iris and the tubercular mass continuous with it in the cornea. Some giant-cells with surrounding reticular area are indicated in the growth. × 9.

Plate I.

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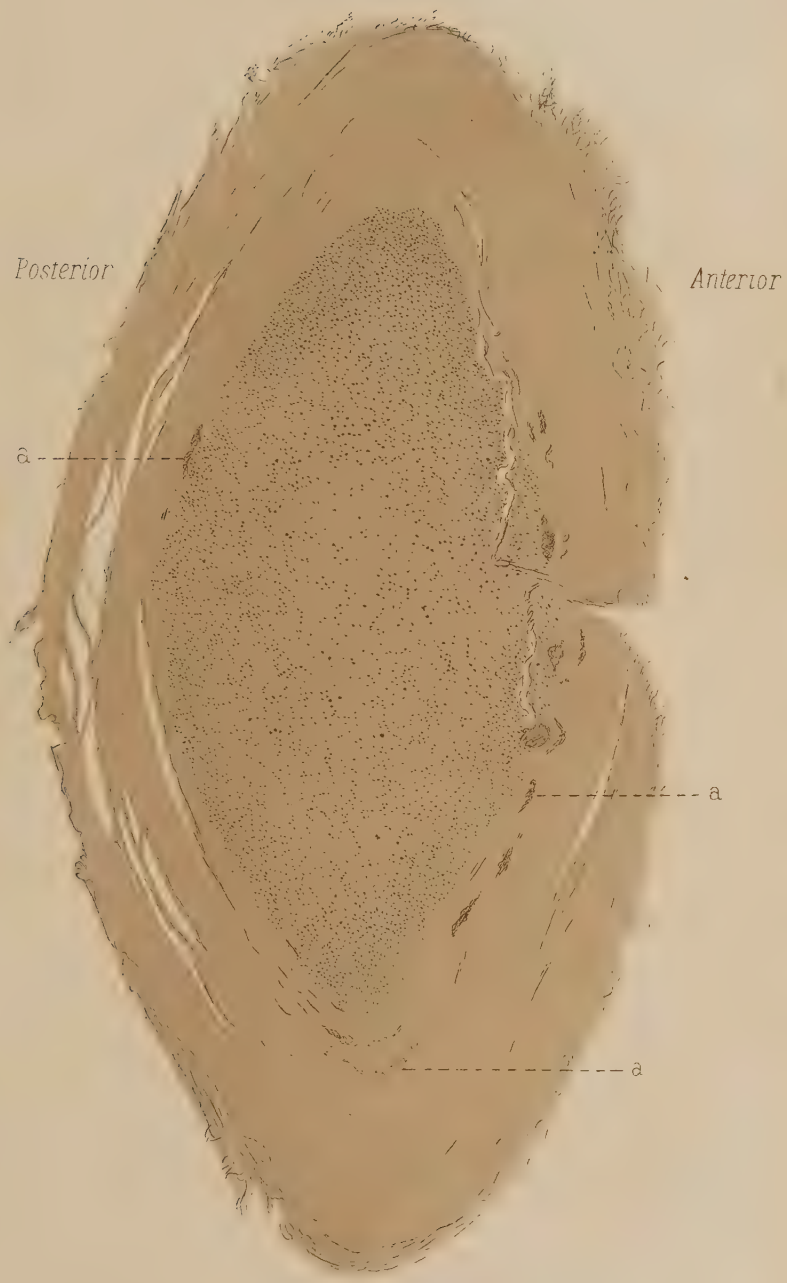

M.H. Lapidge del. & lith.

Danielsson & Co, imp.

Fig. 1.

Fig. 2.

M. H. Lapidge del. & lith. Danielsson & Co, imp.

THE CAPSULO-PUPILLARY MEMBRANE, WITH SOME VARIETIES OF ITS PERSISTENCE.

By W. J. Collins, M.S., M.D., B.Sc. (Lond.)

In vol. xi of the Ophthalmic Hospital Reports I detailed and figured a case of persistent capsulo-pupillary membrane. Such cases are, I believe, not so rare as the statements in some text-books assert, and the absence of any reference to them in others would imply. Neither do I find in most modern English ophthalmic works any distinction made between the pupillary membrane and the capsulo-pupillary membrane; instances of persistence of the latter being described under the name of the former, whereas it would be more in accordance with embryological observation to regard the pupillary membrane as merely a specialised portion of the capsulo-pupillary envelope. Moreover, there is a very great variety in the disposition of the vestigial filaments of these membranes, which the following cases which I have myself observed will suffice to exhibit:—

Case I. Caroline De C., æt. 72, came complaining of failing sight, and had striæ in both lenses; but oblique illumination revealed in each eye delicate brown radial threads, of the same hue as the iris, proceeding from the periphery of the *circulus minor iridis* or *corona*, in the lower and inner quadrant to the lens capsule a little short of the anterior pole (*vide* Fig. 1). She had always had good sight, was admired in her youth for her "*beaux yeux*," and never remembers any inflammatory attack. The symmetry, situation, colour, and disposition of these capsulo-pupillary threads, corroborated by the history, exclude their possible confusion with synechiæ consecutive to iritis. This was an indubitable instance of persistent capsulo-pupillary membrane. I have notes of six cases of this variety.

Case II. John G., æt. 9, came to hospital on account of catarrhal ophthalmia. On examining his left eye there were

seen four or more fine fibrils stretching chord-like across the lower half of the pupil, entirely unconnected with the lens-capsule, and attached peripherally to projecting tufts in the *corona* between the circles of the iris (*vide* Fig. 2). The other eye was normal. This was a case of persistent pupillary membrane proper. I have notes of eight cases of this kind, and must have seen many more.

Case III. Mary R., æt. 17, a patient of Mr. Tweedy's, who kindly allowed me to see the case, applied at hospital on account of a white speck in the left pupil which she was under the impression had only appeared during the previous fortnight. The white speck under oblique illumination and magnification turned out to be a milky-white circumscribed plaque about 2 mm. by 1 mm. with the long axis vertical, whence proceeded four radial blue-grey filaments to be attached to the coronal tufts of the iris as in the preceding case (*vide* Fig. 3). Vision was unimpaired; there was no history of inflammatory attack; the other eye was normal. This case corresponds closely with the one I figured on page 346 in vol. xi of these Transactions in a woman æt. 21; except that in this instance the capsular opacity at the site of insertion of the filaments was larger and had attracted attention (*vide* Fig. 7).

Case IV. James K., æt. 12, came to the North-West London Hospital on June 27 last on account of a blow on the left eye and subsequent failure of vision; the eye exhibited compound myopic astigmatism, and with glasses he read $\frac{6}{9}$, but on examination with oblique illumination I found a variety of persistent pupillary membrane which I had not previously encountered. Three threads attached to the corona of the iris were to be seen, one arising where another was inserted, and none of them encroaching upon the pupillary area (until the pupil was dilated with atropine), but passing tangentially to it around the lower and inner parts of its circumference (Fig. 4).

Case V. Jessie C., æt. 12, came on account of an injury to the left eye of a trivial character, but exhibited yet another variety of vestige. Proceeding from one of the coronal tufts

of the iris at the upper and inner part of it was a delicate lash about $1\frac{2}{3}$ mm. in length and 1 mm. in breadth at its iridic origin, and fining down to almost imperceptible tenuity at the other end, which floated free in the anterior chamber (*vide* Fig. 5). This I take to be an instance of a persistent filament of the pupillary membrane, the other insertion of which had either been ruptured by pupillary movements, or had disappeared by that process of obsolescence prior to birth which normally occurs, and which this individual filament had escaped. I have seen two other examples of this same variety, one in a well-known gentleman of 55, another in a boy aged 3.

Case VI. Joseph S., æt. 54, complained of recently noticed dimness of vision; smoked half an ounce of shag a day; had had a primary sore 30 years ago, but never had any inflammatory attack in his eyes. V. R. $\frac{6}{18}$, L. $\frac{6}{9}$, unimproved by glasses. Vitreous hazy, and some neuro-retinitis. Both his eyes exhibited yet another peculiarity, which I think is due to persistence of a portion of the vascular pupillary membrane which usually makes itself scarce. In his eyes that part of the iris which I have alluded to as the *corona* which marks the junction of the *circulus major iridis* with the *circulus minor* is greatly exaggerated, the tags and tufts, presumably vascular in structure, overlapping the circulus minor and forming an irregular frilled margin to the pupil (Fig. 6). On casual inspection they had the appearance of multiple synechiæ amounting to exclusion of the pupil, but the motility of the iris and the complete dilatation which atropine effected negatived this suggestion. This is the only example of this variety which I have seen.

The literature upon the subject of this communication is not very abundant, is mostly modern, and is very much scattered. Ancient authors do not appear to mention it at all. Wrisberg, Wardrop, and Beer cite instances.

Cohn* of Breslau has paid considerable attention to the subject, and has figured, at any rate, three varieties,

* Cohn. Klinische Monatsblätter für Augenheilkunde, 1867, pp. 62–72.

all of which, however, he describes as persistence of the pupillary membrane, though in two of his cases there is distinct capsular attachment (*vide* Fig. 8).* Weber, Alfred Graefe, Horner, and Mooren have also recorded cases. I can find no mention of the condition in Mackenzie.† Stellwag‡ refers to the capsulo-pupillary membrane in the anatomical part of his work, but gives no instance of its persistence after birth. The modern English text-books of Juler, Lawson, Swanzy, and Nettleship§ do not distinctively refer to persistence of the capsulo-pupillary membrane, though the last insists on the importance of diagnosing such vestiges from synechiæ. In Landolt and Wecker's Traité Complet‖ there is an excellent and clear account of the capsulo-pupillary membrane and of cases of its persistence.

The explanation of the occurrences which lead up to such cases as I have noted above appears to be as follows:—The deeper portion of the vascular mesoblastic intrusion which takes place after the formation of the secondary optic vesicle and between this and the epiblastic lens, surrounds the latter with a complete tunic, and obtains its blood from the hyaloid artery, a branch of the arteria centralis retinæ. When the iris, later on, sprouts from the choroid, and its anterior margin advances centripetally, leaving at first coloboma and pupil, and, lastly, pupil only, a portion of the vascular tunic of the lens or capsulo-pupillary membrane is intercepted by the iris and lies in front of it, while their vessels anastomosing effect a junction between the anterior surface of the iris at the *corona* and the intercepted portion which is henceforth the pupillary membrane. Posteriorly this membrane retains for a time its connection with the rest of the lens tunic, which latter when no longer vascularised by the hyaloid

* Cohn. Klinische Monatsblätter für Augenheilkunde, 1867, pp. 62–72. Case 1 and Case 2.

† "A Practical Treatise on Diseases of the Eye," 4th edit,, 1854.

‡ "Treatise on Diseases of the Eye," 1868, under Cataract.

§ Nettleship. "Students' Guide to Diseases of the Eye," 1884, p. 124.

‖ "Traité Complet d'Ophthalmologie," 1886, tome ii, p. 381.

artery fades into the capsule of the lens. It is easy to see how by the failure in one or other of these successive steps, by which the final condition which we recognise as normal is arrived at, the different vestigial anomalies referred to above may be determined. Proceeding from behind forwards we may have (1) a persistent hyaloid artery or patent canal of Stilling; (2) some opacities occasionally seen in children in the hyaloid fossa may not improbably be due to the retention of, or opacification of, or failure to clarify of, some portion of the posterior part of the capsulo-pupillary tunic; (3) some instances of circumscribed opaque plaques on the anterior surface of the lens, as in Case III, and as figured in some of Cohn's cases (Fig. 8) either with or without connecting threads with the iris; possibly, too, some of those cases of anomalous juvenile capsular cataract, and of otherwise inexplicable anterior polar cataracts, may be referable to persistent and more or less altered portions of this temporary vascular membrane; (4) we have the condition, as in Cases I and III, in which that portion of the membrane proceeding from the lens to the iris is retained; (5) then the class of cases to which the name of persistent pupillary membrane is truly applicable (Case II), where the threads are attached to the iris only, passing diametrically or chordwise across the pupil, or as in Case IV, only touching it tangentially; (6) lastly, the vascular connections established between the anterior surface of the iris and the portion of the capsulo-pupillary membrane which is intercepted by it, may alone persist, either in part (as in Case V) or in entirety (as in Case VI).

The diagnosis of these cases is important as well as merely interesting. Cahsulo-pupillary tags are frequently mistaken for synechiæ due to iritis, and the patient's character is unjustly impugned, while his constitution is unnecessarily assailed with vigorous medication.

As regards treatment, it is seldom called for; sight being often but not always unimpaired; operative pro-

cedure would in most cases be accompanied with the risk of injuring the lens. In Case I, where senile cataract has supervened, it is possible that the capsulo-pupillary adhesions which exist would not be less unfavourable impediments to successful extraction than ordinary iritic synechiæ.

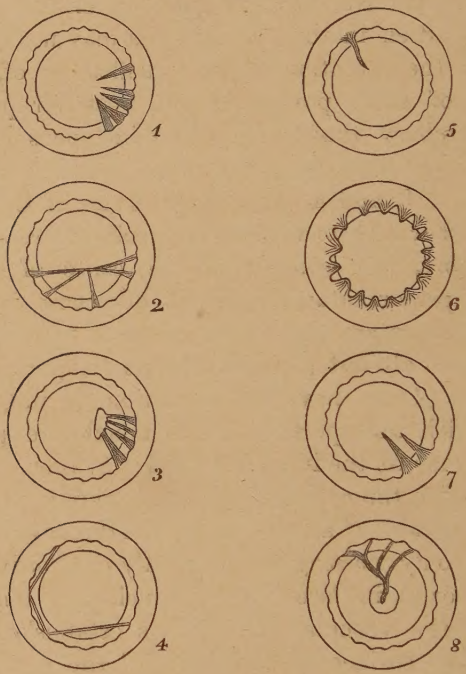

Fig. 1. Case of persistent capsulo-pupillary membrane.
„ 2. Case of persistent pupillary membrane.
„ 3. Case of persistent capsulo-pupillary membrane with white plaque on capsule.
„ 4. Case of persistent pupillary membrane with tangential filaments.
„ 5. Case of persistent pupillary membrane represented by a single lash.
„ 6. Case of persistent pupillary membrane in form of excessive corona.
„ 7. Case of persistent capsulo-pupillary membrane.
„ 8. Case of persistent capsulo-pupillary membrane with central capsular opacity. After Cohn.